Dissertation on

MERITS AND DEMERITS OF SCLEROTHERAPY IN THE MANAGEMENT OF GRADE I AND II HEMORRHOIDS. A STUDY OF 100 CASES

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

for the award of degree of

M.S. (BRANCH - I)

GENERAL SURGERY

GOVERNMENT STANLEY MEDICAL

COLLEGE & HOSPITAL

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU

APRIL – 2013

CERTIFICATE

This is to certify that this dissertation entitled **"MERITS AND DEMERITS OF SCLEROTHERAPY IN THE MANAGEMENT OF GRADE I AND II HEMORRHOIDS"** is bonafide record work done by **Dr. ABDUL MAJID JAFFAR SATHIQ** under my direct supervision and guidance, submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of University regulation for M.S., Branch I – General Surgery Degree Examination to be held on April 2013.

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DECLARATION

I **Dr. ABDUL MAJID JAFFAR SATHIQ** solemnly declare that this dissertation titled **"MERITS AND DEMERITS OF SCLEROTHERAPY IN THE MANAGEMENT OF GRADE I AND II HEMORRHOIDS"** has been done by me.

I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Surgery degree Branch –I (General Surgery) to be held in April - 2013.

Dr. ABDUL MAJID JAFFAR SATHIQ

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CONTENTS

INTRODUCTION

Although few people have died of hemorrhoidal disease, many patients wish they had, particularly after therapy, and this fact led to the beatification of St. Fiachre, the patron saint of gardeners and hemorrhoidal sufferers. Hemorrhoids means blood flowing (Greek: Haema=blood and Rhoos=flowing). The latin word 'pila' from which the word 'pile' is derived, actually means a ball. Since ancient times haemorrhoids have been the most disturbing diseases. Vascular structures in anal canal which help in stool control are called haemorrhoids. They are composed of arterio-venous channels and connective tissue, acting like a cushion in their physiological state. In clinical practice of general surgery, it is the most frequent problem which the surgeons have been treating for centuries. These swollen inflamed veins cause bleeding, discomfort, and itching in the anus or lower rectum. If the symptoms are minimal, patients do not always need any treatment. The first recommendation usually is making simple lifestyle changes through diet (increase in fibre intake, to drink plenty of water and to avoid too much caffeine) and exercises. Symptomatic hemorrhoids can be treated both non operatively and operatively. Non operatively, hemorrhoids can be treated by regulating the bowel motility by various proprietary creams inserted into the rectum from a

collapsible tube fitted with a nozzle with a hip bath or by using hydrophilic colloids (isogel, dulcolax, lactulose etc.). Operative management includes injection sclerotherapy, cryosurgery, elastic rubber band ligation to the base of each haemorrhoid, laser therapy, infra red photo coagulation and various formal surgeries. Though various modalities have been described in managing them effectively, it recurs unless the causative factor have been eliminated. But the cause of hemorrhoids remains unknown.[1] Factors which increase intra abdominal pressure, particularly constipation, play an important role in their development. The operative technique for hemorrhoidectomies described by Milligan Morgan and Ferguson and their modified ones are the most effective and widely used haemorrhoid treatment modalities.[2] These techniques are dealt with many complications like post operative pain, bleeding, sphincter dysfunction etc. thereby increasing the post-operative morbidity and delay in returning to their work. Moreover these techniques are less applicable for elderly malnourished, patients with severe anaemia and for those having additional co-morbid factors like ischemic heart diseases.

Injection sclerotherapy is one of the oldest techniques used for treating haemorrhoids. It is a procedure of injecting 2-5ml of sclerosant in the submucosal plane of dilated hemorrhoidal vein under local

anaesthesia by direct visual examination, causing inflammation and scarring leading to tissue necrosis, where the dilated vein shrinks, collapses and hardens in a period of time thereby cutting off the blood supply to the hemorrhoids. Injection treatment is ideal for first degree internal bleeding hemorrhoids. Curative treatment can be given even for early second degree hemorrhoids but they often relapse requiring multiple injections. Grade III and IV hemorrhoids or those with noticeable prolapse do not respond well to this procedure. Advantages of injection sclerotherapy are that they are relatively painless, less time consuming, patients soon return to work, complications are minor and the curative rate is high.[3,4] Many agents can be used as sclerosants like 5% Phenol in almond oil or vegetable oil, 2% sodium tetradecyl sulphate, quinine etc.

In this study, 100 patients where studied by treating grade I and II hemorrhoids using 2% sodium tetradecyl sulphate and their ymptomatologies where compared pre-operatively and post-operatively by following them up for a period of one year. Some of them required repeated injections and a few ended up in failure of the treatment. These results where dependent on the grade of hemorrhoid at the time of presentation. Patients with heart diseases, severe anemia and malnutrition, coexistent liver disorders etc., presenting with grade III

hemorrhoids were also tried by treating them using sclerotherapy. But their results were not found to be satisfactory, though there was conversion from grade III haemorrhoids to lower grades in a few, lasting only for a short period of time. Moreover these patients were found to land up in complications easily than those patients without comorbid factors presenting with grade I and II hemorrhoids.

Thus injection sclerotherapy though ideal for grade I hemorrhoids is a temporary modality of control for patients presenting with higher grades and for those in whom definitive operative procedure cannot be undertaken. Though it is one of the ancient treatment, this technique is to be considered by all general surgeons due to its benefits of cost effectiveness, treatment on outpatient basis, ease of administration and early return to work.

AIM OF THE STUDY

1. To compare the preoperative and postoperative symptomatology of patients presenting with grade I and II hemorrhoids after treating them with injection sclerotherapy.
2. To reduce the postoperative morbidity of patients presenting with hemorrhoids rendering them early return to work, less postoperative complications and avoiding period of stay in the hospital.
3. A conservative approach to ischemic heart disease, severely malnourished and anemic patients presenting with hemorrhoids using injection sclerotherapy.

HISTORY

Hemorrhoidal disease has been known since Egyptian Papyrus dating back to 1700BC.

Immediate recovery in olden days was obtained by placing fine linen strip over anus which was smeared previously with ground, titrated and cooked leaves of Acacia.

Similar to modern rubber-band ligation, the Hippocratic corpus has practiced it in the management of hemorrhoids dating back to 460BC.

The complications of ligation and excision procedure and the surgical techniques was described by Celsus (25BC – AD15).

The surgical techniques were developed by European surgeons like Guy de Chauliac, Lanfranc of Milan, John of Ardene and Henri de Mondeville in thirteenth century.

In 1398 the word ‘hemorrhoid’ was first used in English.

In 1682, D Zollikofer was the first to report sclerotherapy by injecting them into the veins and inducing thrombus formation.

In 1853 Debout and Cassaignaic by injecting perchlorates of iron successfully treated varicose veins.

By injecting tannin and iodine into veins, 16 cases of varicose veins was cured by Desgranges in1854.

Sclerotherapy of symptomatic internal hemorrhoidal disease was first advocated by Mitchell in 1871 and has enjoyed significant experience.

But due to the adverse effects of the drug it was abandoned. The introduction of sclerosants like sodium tetradecyl sulphate has marked the beginning once again making its use in many conditions like oesophageal varices, varicose veins as well as in the management of grade I and II hemorrhoids. Though various procedures of managing hemorrhoids like rubber band ligation, cryotherapy and laser therapy are available, injection sclerotherapy is comparatively easier, does not need much of a surgical expertise, relatively cheaper and its results have made this approach as one of the best conservative procedures available.

ANATOMICAL CONSIDERATIONS

ANAL CANAL: The anal canal is 3cms (over an inch) long approximately. Like the rest of the gut it is a tube of muscle, but the fibres are all circular, consisting of internal (smooth) and external (striated) sphincters. These sphincters hold it continually closed except for the temporary passage of faeces and flatus. The junction of rectum and anal canal is at the pelvis floor, i.e., at the level where the pubo-rectalis muscle clasps the gut and angles it forward. From this right angled junction with the rectum the anal canal passes downwards and somewhat backwards to the skin of the perineum. Anatomists consider the anal canal to begin at the dentate line and end at the anal verge. However, most surgeons consider the anal canal to start at the anorectal ring and terminate at the anal verge.

Just above the dentate line, the rectal mucosa forms from 8–14 longitudinal folds known as the rectal columns. Between each two columns at the dentate line is a small pocket termed an anal crypt. Small, rudimentary anal glands open into some, but not all, of these anal crypts. The glands may extend through the internal sphincter as far as the intersphincteric plane, but they do not extend into the external sphincter.

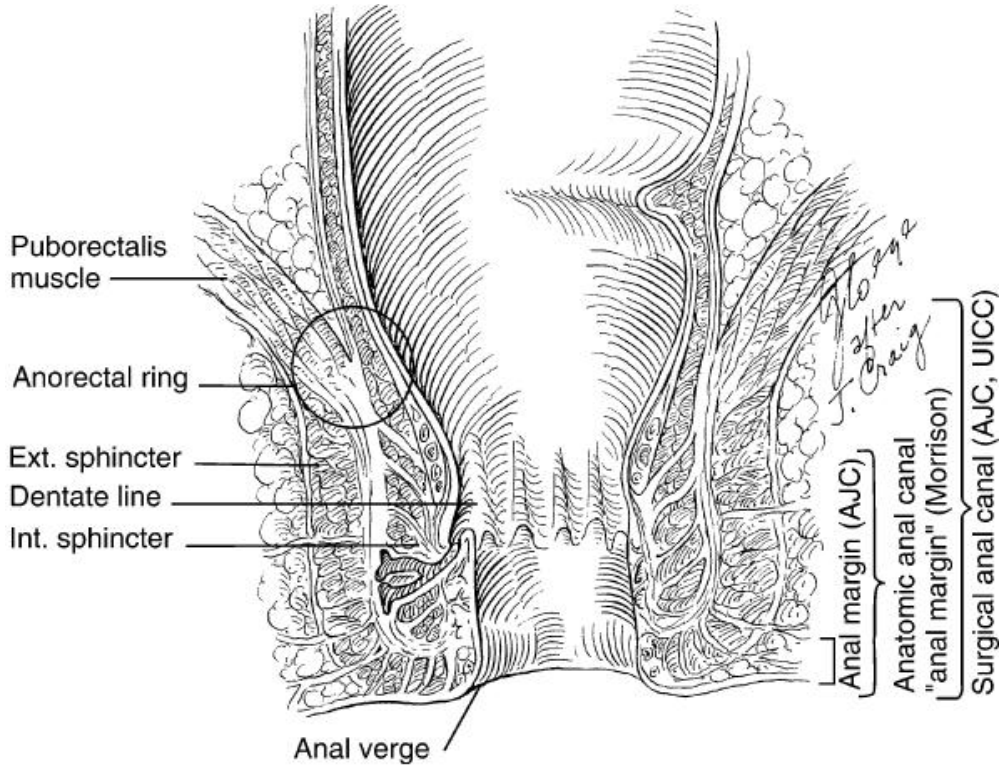


Fig - 1 Anatomy of the anal canal and margin.

THE LINING OF THE ANAL CANAL: The lining of anal canal transitions from normal, squamous, hair-bearing skin to gastrointestinal columnar epithelium incrementally, between the anal verge and top of anal canal in a short distance. The anal canal is lined with mucous membrane in its upper two-thirds and skin in its lower one-third. The junction of the two is abrupt; it is at Hilton's white line. This line is the site of attachment of the fascia derived from the longitudinal muscle coat of the rectum and it is a watershed dividing upper and lower

zones of arterial supply and venous and lymphatic return. It also separates two zones of different nerve supply. It is not ‘white’ on inspection, but was so described by Hilton on account of its relative avascularity. The part above Hilton’s line (mucous membrane) is derived from the endoderm of cloaca, while the part below the line (skin) is derived from the ectoderm of anal pit or proctodaeum.

The cloacal part, is lined with typical large-gut mucous membrane containing mucous crypts and covered with columnar epithelium (goblet cells). There are mucous glands in the submucosa. Occasionally in the lower part, between the annulus hemorrhoidalis and Hilton’s line, there are no mucous crypts and the epithelium is in several layers. This is the zone of so-called ‘transitional’ epithelium; it is not, however, the urine-proof transitional epithelium of the urinary passages, but a thinned out epidermis extending up from the anal part. It is also called as anal transition zone where the squamous and columnar comingle comingle at the level of dentate line. It is a modified squamous stratified epithelium. This zone is called pectin by clinicians.

The anal pit part is lined with thin hairless skin without skin appendages. It is surfaced with squamous stratified epithelium.

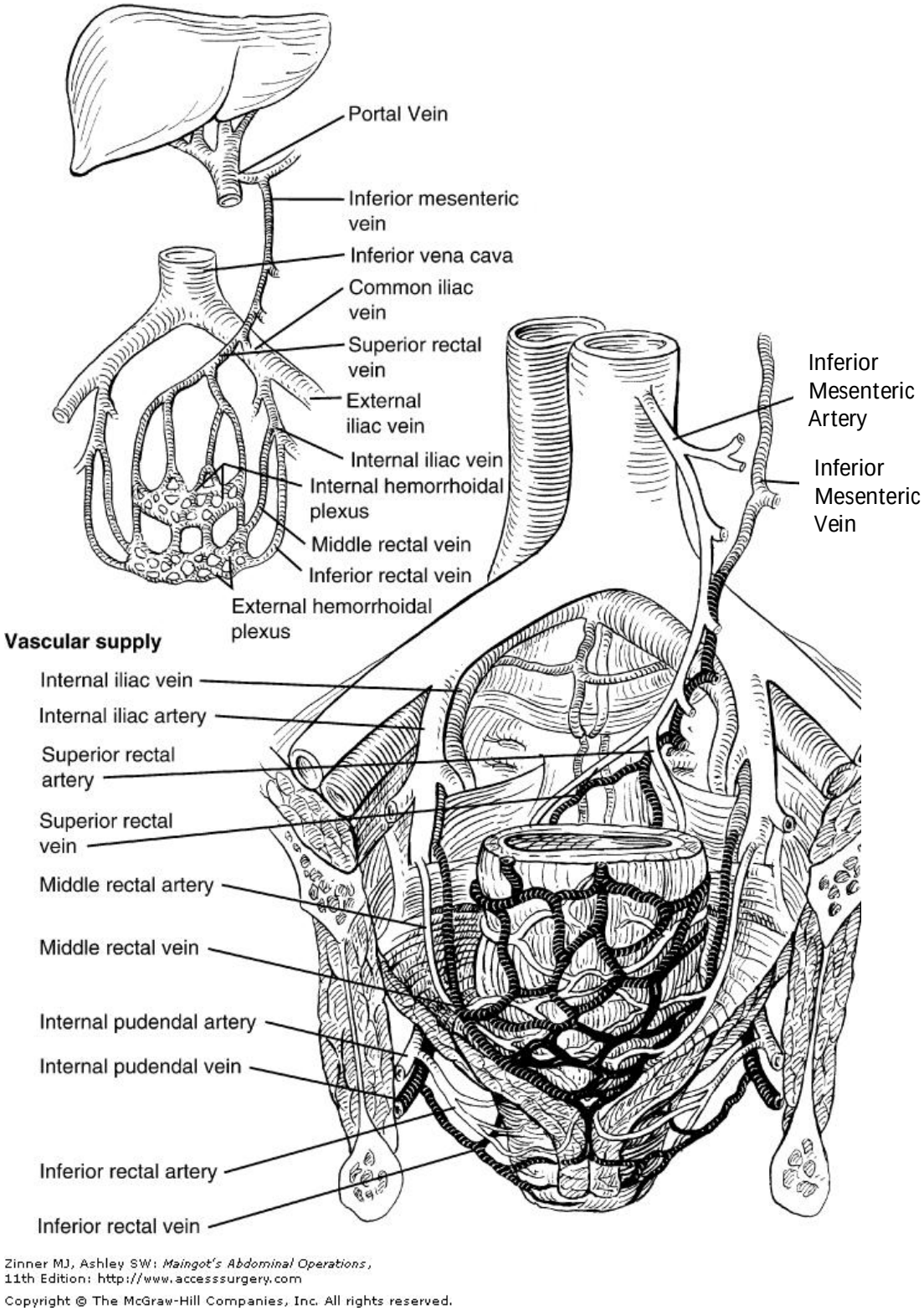
Hidradenitis suppurativa leading to fistula formation can arise from appendages of skin, hence can only be seen distal to dentate line

outside the anal verge whereas fistulas resulting from cryptoglandular disease and Crohn's are seen proximal to the dentate line. This histology is also helpful in understanding the behaviour, management and origin of cancers. Squamous cell carcinomas arising from anal skin tend to occur below dentate line and are treated by wide excision followed by chemoradiation, whereas adenocarcinomas arising from anal canal and rectum are seen above the dentate line and are treated by surgical removal of rectum followed by adjuvant chemoradiotherapy.

The Pectinate Line and Changes in the Surgical Anal Canal		
	Below the Pectinate Line	**Above the Pectinate Line**
Embryonic origin	Ectoderm	Endoderm
Anatomy		
Lining	Stratified squamous	Simple columnar
Arterial supply	Inferior rectal artery	Superior rectal artery
Venous drainage	Systemic, by way of inferior rectal vein	Portal, by way of superior rectal vein
Lymphatic drainage	To inguinal nodes	To pelvic and lumbar nodes
Nerve supply	Inferior rectal nerves (somatic)	Autonomic fibers (visceral)
Physiology	Excellent sensation	Sensation quickly diminishes
Pathology		
Cancer	Squamous cell carcinoma	Adenocarcinoma
Varices	External haemorrhoids	Internal haemorrhoids

The arterial supply of the cloacal part is derived from the superior rectal artery (the artery of hind gut) as far as the white line, where the hind gut ends. The superior rectal artery, the terminal branch of the inferior mesenteric artery, descends to the upper rectum where it divides into lateral branches. Subsequent smaller divisions penetrate the rectal wall. The middle rectal arteries arise from the internal iliac arteries and supply the distal rectum and upper anal canal. The inferior rectal arteries, branches from the internal pudendal arteries, cross the ischiorectal fossae to supply the anal sphincters. The lower third (skin) below Hilton's line, is supplied by the inferior rectal artery (which also supplies the sphincters). These arteries do not anastomose with each other.

The veins of the upper part (mucous membrane) drains upwards into the submucous plexus of the ampulla of the rectum. The veins of the skin part, below Hilton's line, drain downwards into the inferior rectal vein or into tributaries of the saphenous vein. They do not communicate with the veins of the cloacal part. Hilton's line is a venous watershed.



Zinner MJ, Ashley SW: *Maingot's Abdominal Operations*, 11th Edition: http://www.accesssurgery.com

Fig – 2 Vascular supply of the anus and rectum. Blood returns from the anus via two routes. Below the dentate line, the external hemorrhoidal plexus drains into the inferior vena cava via inferior pudendal veins. Above the dentate line, the internal hemorrhoidal plexus drains into the portal system via the superior rectal vein.

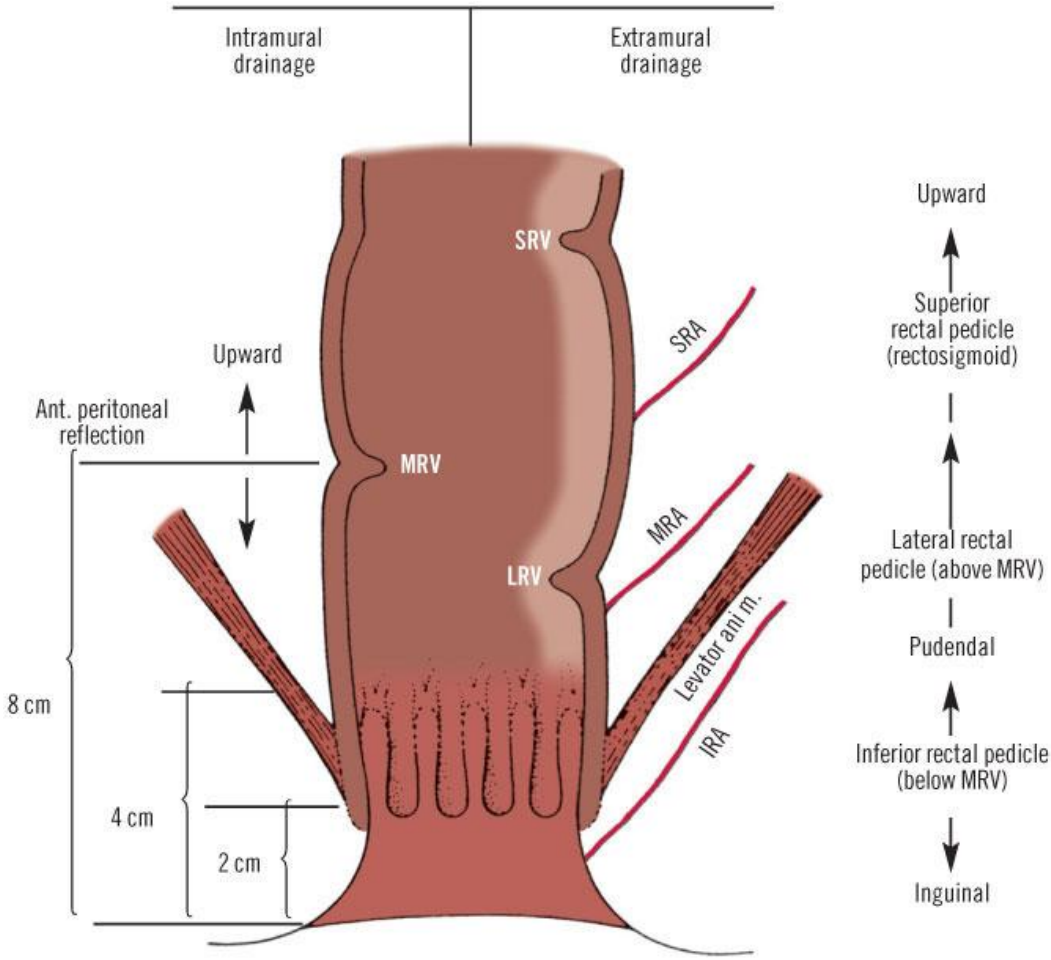


Fig – 3 Diagram of lymph drainage of the anus and rectum. The watershed for extramural drainage is at the pectinate line. The watershed for intramural drainage is at the level of the middle rectal valve, about 8 cm above the anal verge. IRA, Inferior rectal artery. MRA, Middle rectal artery. SRA, Superior rectal artery. LRV, Lower rectal valve. MRV, Middle rectal valve. SRV, Superior rectal valve.

The lymphatics of the cloacal part pass upwards from lymphatic follicles in the mucous membrane to join those of the rectum. Those of the skin part drain with the rest of the perineum into the medial group of superficial inguinal nodes.

The nerves of the cloacal part are autonomic, from the inferior hypogastric plexuses, and this part of the mucous membrane is relatively insensitive to touch though it registers pressure (e.g., of the examining finger or a rectal tube) and it can distinguish between faeces and flatus. The nerves of the skin part are somatic, from the inferior rectal nerve and the skin is highly sensitive (e.g., to the pain of fissure in ano). Below the dentate line, cutaneous sensations of heat, cold, touch, and pain are conveyed by afferent fibers in the inferior rectal nerves. Cephalad to the dentate line, poorly defined dull sensations, elicited when the mucosa is pinched or internal hemorrhoids are ligated, are probably carried by parasympathetic fibers. This helps us in understanding why external hemorrhoids is painful and internal hemorrhoids presents as painless bleeding.

VEINS OF THE ANAL CANAL: The mucous membrane of the upper two-thirds contain a rich plexus of veins. These drain upwards by vertical channels which, when full of blood, raise ridges in the mucous membrane known as anal columns. Three of these veins, situated at 3, 7 and 11 o'clock position when the patient is viewed in the lithotomy position, are apt to become varicose as the three 'primary' haemorrhoids. The veins pass up in the submucosa to join the submucous plexus in the ampulla of the rectum, where the blood is

diverted to either the portal or the systemic systems. The anal columns are joined by cross channels of anastomosing veins which raise small mucosal folds known as anal ‘valves’. These anastomosing cross channels form a venous ring known as annulus hemorrhoidalis, or zona hemorrhoidalis. The situation of this dentate ring of valves is variable. It often lies low in the anal canal, close to the mucocutaneous junction (Hilton’s line), but wherever it may be it is always in the large bowel (cloacal) part of the anal canal. It never lies in the junctional part, and it is wrong to imagine that it represents the site of the cloacal membrane of the embryo. The anal columns vary in prominence according to the amount of their contained blood. The anal valves remain constant irrespective of the amount of blood in the annulus. The tiny curved ridges of mucous membrane produce pockets above them; these may become infected.

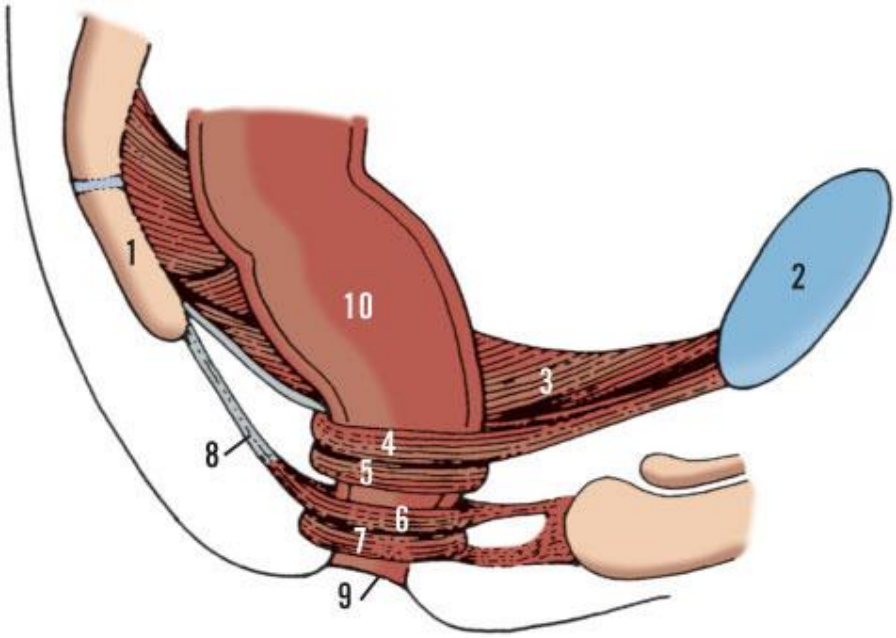


Fig – 4 Diagram of the extrinsic muscles of the surgical anal canal. 1, Coccyx. 2, Pubis. 3, Levator ani muscle. 4, Puborectalis muscle. 5, Deep external sphincter. 6, Superficial external sphincter. 7, Subcutaneous external sphincter. 8, Anococcygeal ligament. 9, Anal verge. 10, Rectum.

THE ANAL SPHINCTERS: The upper border of the anal sphincteric complex is the anorectal ring. It may be palpated by digital examination about 1.0–1.5 cm above the dentate line. The anal canal is always closed except for the passage of flatus or faeces. It is held closed by a sphincteric tube of muscle. The tube is 3cms (over an inch) long, lined as described above. The muscle is in two distinct sphincteric entities, internal and external. Each occupies two-thirds of the canal, so that they overlap at the middle third. The internal sphincter is of smooth muscle and lies around the upper two-thirds of the canal. The external

sphincter is of striated muscle; it clasps the lower part of the internal sphincter and surrounds the lower two-thirds of the canal.

INTERNAL SPHINCTER: This occupies the upper two-thirds of the anal canal, i.e., down to the Hilton's line. It is the thickened lower end of the inner circular muscle of the rectum, with which it is continuous. It is made of smooth muscle. It is innervated from the inferior hypogastric plexuses; sympathetic stimulation contracts the muscle, parasympathetic stimulation relaxes it. It is under a voluntary control similar to that of the smooth muscle of the bladder. The sphincter is relatively weak, and is not competent when acting alone; at least some of the deepest part of the external sphincter is essential for complete continence of flatus and faeces. The internal sphincter is surrounded by a loose and distensible fibrous sheath, which is downward continuation of the longitudinal muscle coat of the rectum.

EXTERNAL SPHINCTER: This tube of striated muscle surrounds the lower two-thirds of the anal canal. It consists of three parts (each a 'ring' of muscle) lying adjacent to each other in series. The three rings lie superficial, middle and deep, but, unfortunately, they are not so named. The middle 'ring' is,very confusingly, named 'superficialis'; the superficial ring is named 'subcutaneous', and the two are separated by a fascial septum. This fascia is downward prolongation

of the longitudinal muscle of the rectum. It invests the internal sphincter, separating it from the surrounding external sphincter, and turn inwards to become attached to Hilton's white line. From the same level a sheet of this peri-anal fascia passes outward to the pudendal canal and separates the peri-anal space from ischio-rectal space. It likewise separates the 'subcutaneous' from the 'superficial' part of the external sphincter.

CORRUGATOR-CUTIS ANI MUSCLE: This small muscle consists of thin slips of smooth muscle fibres which radiate out from Hilton's white line to be attached to the peri-anal skin. It is part of the panniculus carnosus and has nothing to do with the external sphincter. Its contraction shrinks and therefore cleans the peri-anal skin in many quadrupeds; it is an almost functional vestige in man.

SUBCUTANEOUS EXTERNAL SPHINCTER: This is a thick ring of muscle, not attached to bone. It lies immediately beneath the skin and corrugator fibres, and is separated from the superficial external sphincter by fascial attachment to the white line as mentioned above. It is supplied by inferior rectal nerve (S3, 4) and is easily palpable by the examining fingertip.

SUPERFICIAL EXTERNAL SPHINCTER: This is middle of the three parts of the external sphincter. It is an elliptical muscle,

attached to the perineal body anteriorly and to the tip of the coccyx posteriorly. It is supplied by the perineal branch of S4.

DEEP EXTERNAL SPHINCTER: This is an annular muscle, not attached to bone. It encircles the lower part of the internal sphincter. Posteriorly it blends with the embracing loop of the pubo-rectalis muscle, but anteriorly it forms a complete ring which separates from the pubo-rectalis and fills in the space between the two halves of that muscle in front of the recto-anal junction.

It is essential to the continence of flatus and faeces. Posteriorly the whole ano-rectal ring (pubo-rectalis and profundus sphincter) functions, but anteriorly there is only the profundus to maintain continence; hence the danger of anterior lacerations of the anal canal (e.g., obstetric). The profundus part of the external sphincter is supplied by the inferior rectal nerve (S3, 4).

The peri-anal fascia surrounding the internal sphincter is expansile to accommodate the passage of a faecal mass, but it cannot stretch longitudinally. Many of its fibres traverse the subcutaneous sphincter to become attached to peri-anal skin. Thus the longitudinal muscle fibres of the rectum can retract the anal canal proximally over an extruding faecal mass.

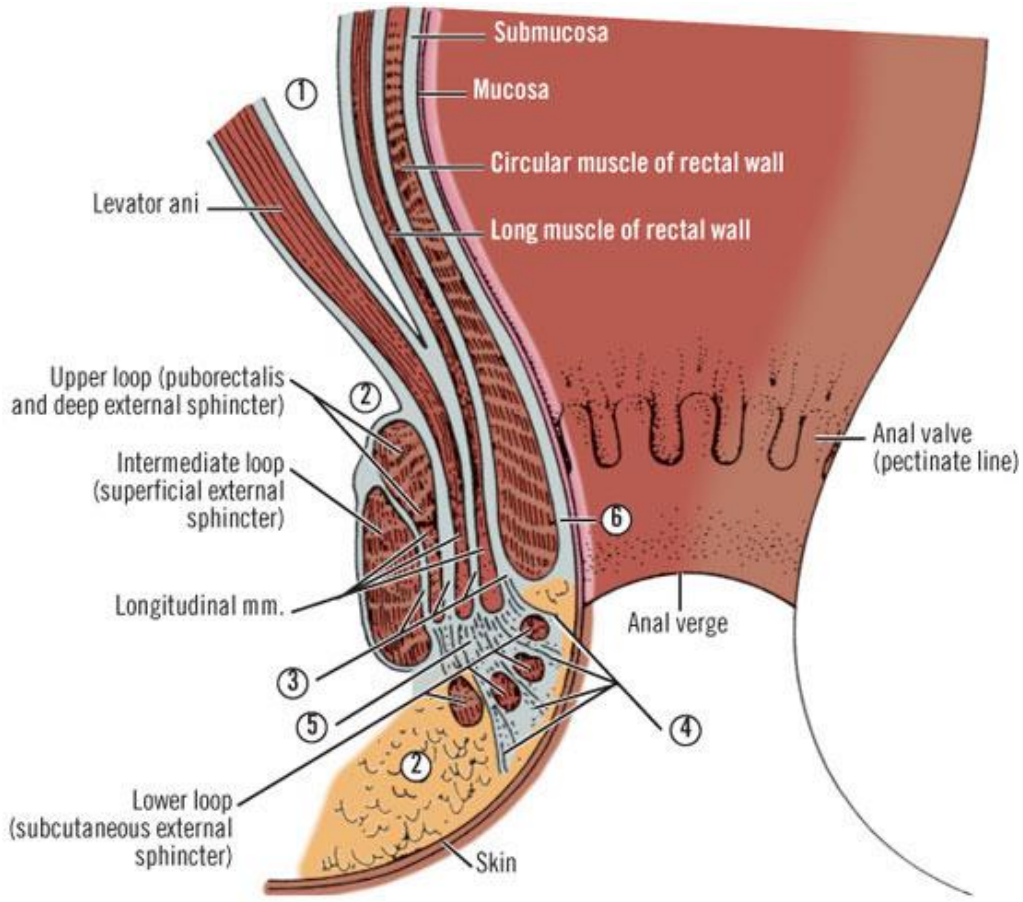


Fig – 5 The spaces of the anus and rectum. 1. Pelvirectal space. 2, Ischioanal (ischiorectal) space. 3, Intersphincteric spaces. 4, Subcutaneous space. 5, Central space. 6, Submucous space.

THE ISCHIO-RECTAL FOSSA: This is a wedge-shaped filling in the lateral part of the anal triangle, and extending forward into the urogenital triangle. It is filled with soft fat which forms 'dead-space' into which the anal canal can expand during defaecation. Its lateral wall is formed by the fascia over the lower part of obturator internus, the falciform margin of the sacro-tuberous ligament, and the tuber ischii. Medially the two fossae are separated by the perineal body, the anal

canal and the ano-coccygeal body, and they are roofed in by the downward sloping levator ani muscles of the pelvic floor.

The junction of the rectum and anal canal is slung in the pubo-rectalis muscle; the rectum is wholly in the pelvic cavity. The name ischio-rectal fossa is a misnomer, and the inferior rectal vessels and nerves supply no rectum but only the anal canal, peri-anal space and skin.

ANO-COCCYGEAL BODY: The fibres of the ilio-coccygeus and of the pubo-coccygeus interdigitate in front of the coccyx in a midline raphe which extends from the tip of the coccyx to the ano-rectal junction. Raphe and skin diverge from each other as they pass forwards to the upper and lower ends of the anal canal. In the midline space between them is a fibro-muscular mass of tissue called the ano-coccygeal body, which separates the two ischio-rectal fossae behind the anal canal. Fibres of the intermediate (superficialis) part of the external sphincter traverse the ano-coccygeal body to become attached to the tip of the coccyx. Lateral to this the sacro –tuberous ligament limits the fossa.

PERI-ANAL SPACE: A prolongation from the longitudinal muscle of the rectum passes downwards as a fascial membrane which splits and is inserted into Hiltons white line internally. An outer

prolongation reaches the side wall of the pelvis, at the pudendal canal. This delicate membrane, the peri-anal fascia, separates the depths of the ischio-rectal fossa from a shallow subcutaneous peri-anal space. The fat in the latter space is contained in small loculi which are separated by fibrous septa almost complete (contrast the large loculi in the ischio-rectal space separated by very incomplete septa). Hence infection in this space gives rise to considerable tension as swelling occurs, with consequent great pain. The peri-anal space, in other words, consists of ordinary subcutaneous fat, and the peri-anal fascia separates it from the fat in the ischio-rectal space.

THE ISCHIO-RECTAL SPACE: The fat in this space is arranged in large loculi which are but incompletely separated by delicate septa. Infection in this space (by far the greater part of the whole ischio-rectal fossa) can cause swelling without tension and with, therefore, a minimum or absence of pain. The space allows dilatation of the anal canal during defaecation. Although the rectum lies above the pelvic floor it can dilate by pressing the sloping levator ani into the space. Similarly the vagina can dilate into the space, especially during parturirion, when the passage of the foetal head obliterates the space.

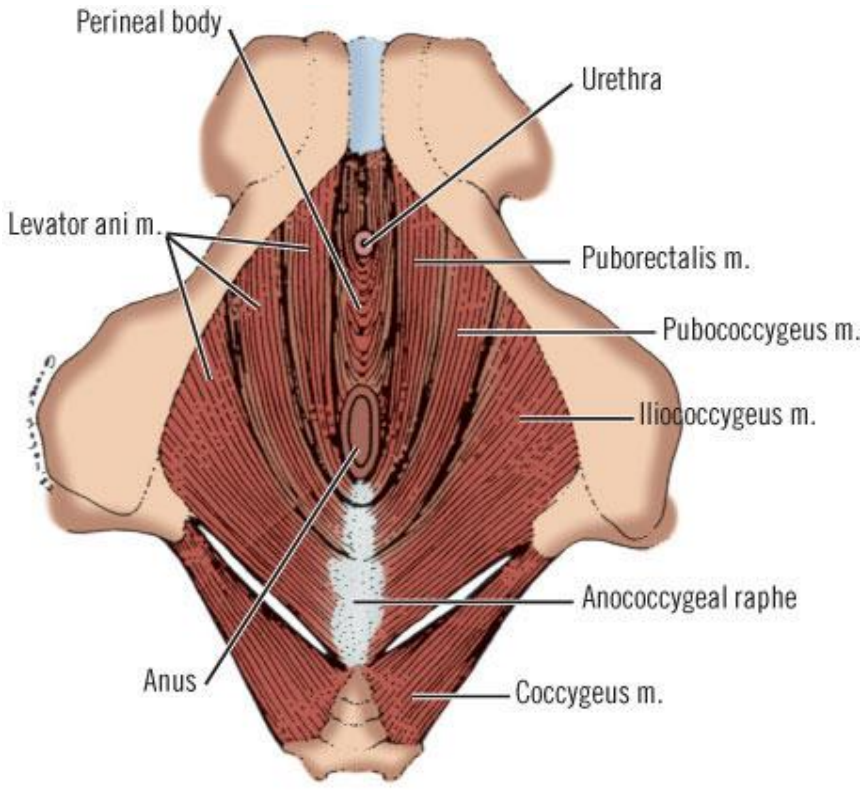
PUDENDAL CANAL: The sacro-tuberous ligament is attached to the medial half of the lower part of the tuber ischii. Its upper edge is

prolonged forward on the medial surface of the ischium as falciform ligament. Above the falciform ligament is the dense fascia on the obturator internus. Here lies a fibrous canal containing the internal pudendal vessels and nerve. This canal, the pudendal canal, is formed from the lateral prolongation of the delicate peri-anal fascia, which splits and thickens to enclose the pudendal neuro-vascular bundle. The pudendal canal connects the lesser sciatic foramen to the posterior edge of perineal membrane. The pudendal canal was called Alcock's canal in former times euphoniously.

LUNATE FASCIA: Arching over the upper margin of the ischio-rectal fat, separating it from the areolar tissue on the lower surface of the levator ani muscle, is the lunate fascia. It commences laterally at the pudendal canal and fades out medially over the profundus division of the external sphincter ani muscle. At its anterior extremity it is prolonged forwards to fuse with the areolar tissue on the lower surface of levator ani muscle. This situation is anterior to the posterior end of perineal membrane; thus each ischio-rectal fossa posseses a forward prolongation into the uro-genital triangle on the lateral aspect of the membranous urethra (male) or vagina (female) along the inferior pubic ramus.

THE PERINEAL BODY: Lying in front of the anal canal is a fibro-muscular mass of tissue fixed to, and forming part of, the pelvic floor. It is composed chiefly of the interdigitating fibres of pubo-prostaticus, but is incremented by both transverses perinei muscles and the 'superficial' part of the external anal sphincter. It extends from the level of the pelvic floor to the skin of the perineum, plugging the space between right and left ischio-rectal fossae. It is indispensable to the support of the pelvic viscera.

Fig – 6 Diagram of the pelvic diaphragm from below. Note that the levator ani is composed of three muscles: puborectalis, pubococcygeus, and iliococcygeus.



PATHOPHYSIOLOGY OF ANAL INCONTINENCE:

Mechanical disruption is usually due to obstetric injury, trauma, or fistula disease in which the external muscle is divided or damaged. Neurogenic incontinence is due to stretching of the pudendal nerves during prolonged labor, descent of the perineum and nerve stretch during straining at stool or rectal prolapse, or systemic disease such as multiple sclerosis, scleroderma, or spinal cord injury. Idiopathic incontinence is due to medical disease such as diarrhea in a patient with limited rectal capacity, irritable bowel syndrome, or sedatives which cause poor sensation in the anal canal in patients with no evidence of neurogenic or mechanical incontinence.

Anal Incontinence Etiology		
Mechanical	**Neurogenic**	**Idiopathic**
Obstetric injury	Pudendal nerve stretch	No clear etiology
Fistula disease	Strain	Medical illness
Trauma	Prolonged labor	Irradiation
Iatrogenic	Trauma	Irritable bowel syndrome
Systemic disease		Multiple sclerosis, diabetes mellitus, scleroderma
Diarrheal states		

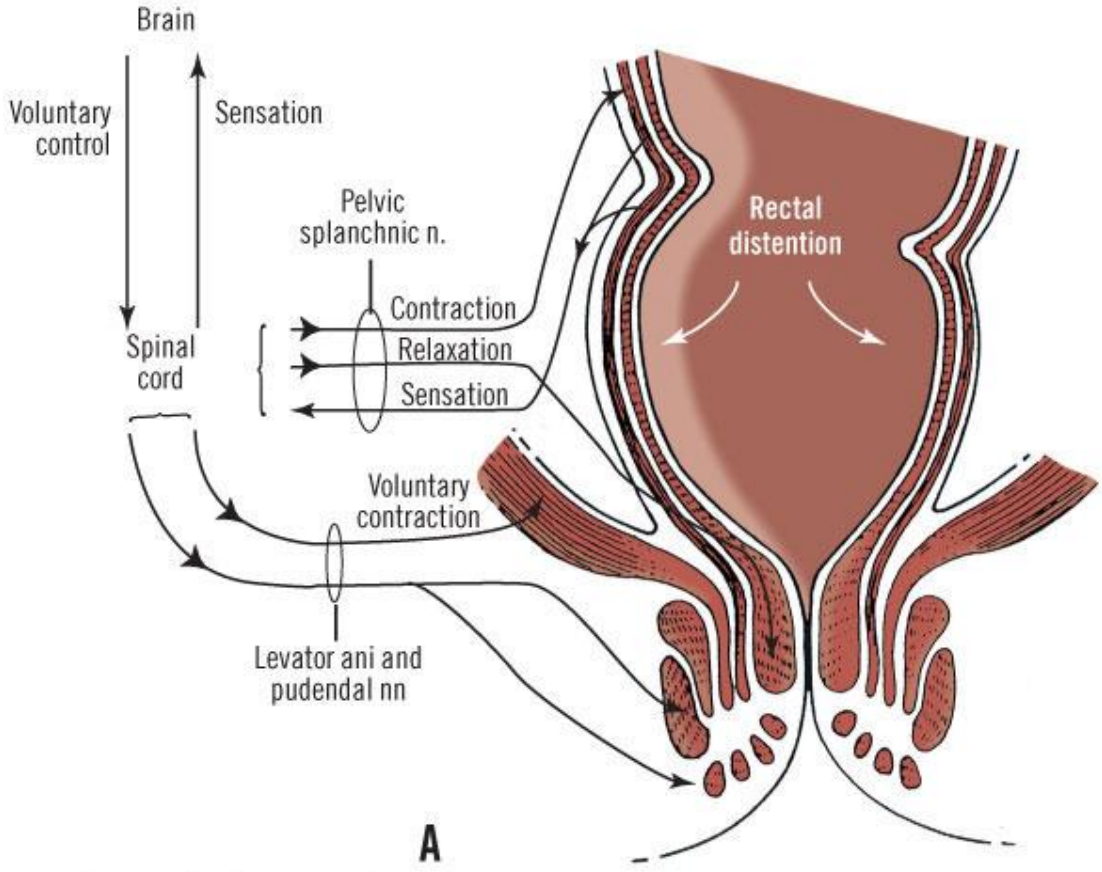
The normal continence mechanism has several components. Rectal capacitance and compliance are essential. The rectum normally holds between 200 and 250 ml. It distends readily with filling and has limited muscular activity intrinsically. The sampling reflex is a function of rectal distension allowing anal sphincter relaxation via an intramural reflex to the internal sphincter. The rectal contents can then be sensed in the sensory nerve–rich transitional zone and anoderm to discriminate the true nature of the rectal contents. This sampling reflex occurs frequently throughout the day to provide continence and also serves to initiate the defecation process. The voluntary external sphincter muscle contraction in response to this sampling reflex provides the final active component of anal continence. The subconscious voluntary contraction of the external sphincter, puborectalis, and pelvic floor muscles provide complete control of rectal contents. The pelvic floor muscles maintain continual activity, even during sleep, to provide anal continence. This also seems to be a learned response since infants and children require 1–2 years to achieve control.

It is important to realize that the degree of incontinence affects the lifestyle of the patient. The frequency of incontinence may vary and the loss of control may involve solid stool, liquid stool, or gas only.

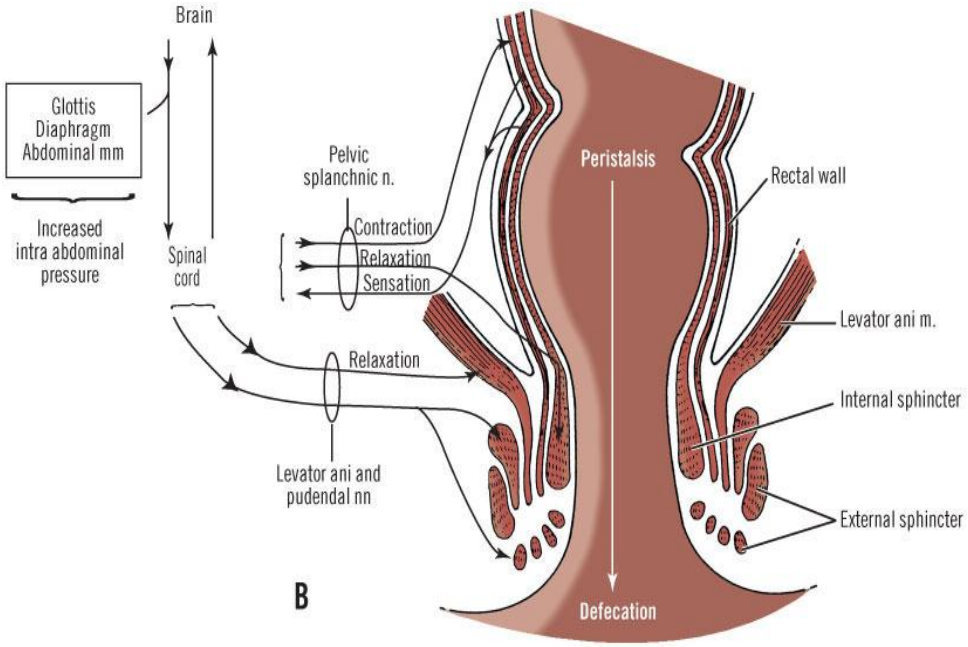
Frequent episodes of incontinence of gas only may be as incapacitating as infrequent episodes of solid stool. It is essential to document the exact type of incontinence before planning treatment. It is especially important to clearly define the incontinence before attempting to report a series of patients who undergo a specific treatment. There is no universally accepted grading scale to assess severity and impact of fecal incontinence. Recently, the American Society of Colon and Rectal Surgeons validated a fecal incontinence severity index and a fecal incontinence quality of life index to help standardize the assessment of anal incontinence.

Fig – 7 Neural pathways involved in defecation are as shown in the diagram below:

A, *Rectal distension initiates relaxation of the internal sphincter and effective voluntary closure by the external sphincter.*



B, *Defecation with relaxation of both sphincters, contraction of muscles in the rectal wall, and increased intraabdominal pressure.*



PHARMACOLOGY

Sodium tetradecyl sulphate is a detergent based anionic surfactant which is a waxy, white solid.

The available concentration ranges from 0.1% - 3%

The mechanism of action is by acting on the lipid molecules of the in the wall of vein causes intimal inflammation and thrombus formation. The resulting fibrosis causes temporary or permanent obliteration of the vein which may be partial or complete.

The recommended dosage varies from 0.5ml to 2ml, ideally 1ml maximum, not exceeding 10ml in single treatment dosage.

It should be stored at a temperature of 20 to 25 degree Celsius.

Its carcinogenic potential is not yet been documented.

Its safety in paediatric patients is not yet established and should be used cautiously by mothers while nursing. Like any other drug it can concentrate in breast milk.

Patients with Buerger's disease (thromboangitis obliterans) and peripheral arteriosclerosis should exercise extreme caution prior to administration of the drug.

It is available as a chemical combination with dibasic sodium phosphate + 0.02ml of benzyl alcohol + anhydrous 4.0mg in water.

The pH is 7.9 which can be adjusted by addition of monobasic sodium phosphate and/or sodium hydroxide.

Adverse reactions include pain, ulceration, discolouration and urticaria at local injection site.

Accidental extravasation can cause sloughing and necrosis of the tissue.

Nausea, vomiting and headache can occur as mild systemic reactions.

It is contraindicated in asthmatic patients as it can cause hay fever, hives, asthma and anaphylactic shock as allergic reactions. So far six deaths have been reported, four of which were due to anaphylactic shock. It is also liable to cause fatal pulmonary embolism.

Sodium tetradecyl sulphate is not advisable for patients taking anti ovulatory agents and should not be mixed with heparin in the same syringe.

It is contraindicated in following conditions:

- Superficial thrombophlebitis

- Allergic and hypersensitivity reactions
- Acute infection with cellulitis
- Large superficial varicose veins communicating with deeper veins via large channels, also in patients with deep vein or valvular incompetence.
- Patients with uncontrolled systemic disease such as tuberculosis, diabetes, neoplasm, sepsis, toxic hyperthyroidism, acute respiratory or skin diseases, blood dyscrasias and asthma.

Many sclerosants are available with different mechanism of actions and relative associated complications. Most common sclerosants are:

- Sodium morrhuate 2% - 5%
- Polidocanal 1% - 5%
- Ethanolamine oleate 5%
- Sodium tetradecyl sulphate

Injecting sclerosant into roof of mouth hardens vibrating soft tissues, hence can be used in the treatment of snoring.

REVIEW OF LITERATURE

Resting anal tone is the pressure generated in anal canal during periods of inattention or sleep to keep it closed. Half of the normal resting anal tone is provided by the internal anal sphincter. The groove between the internal and external sphincter and the lower edge of internal anal sphincter can be palpated 1cm below dentate line approximately. Puborectalis and the external anal sphincter provides the reminder of resting anal tone.

The maximum pressure is generated by voluntary contraction of puborectalis and the external anal sphincter, which is called the maximum voluntary squeeze, can be maintained only for a short period of time. Coughing and sneezing causes reflux contraction of striated sphincteric mechanism. Continence depends on compliance of rectum, interaction between anal sphincters and the type of challenge (liquid, gas or solid). Urgent calls to stools at low rectal volumes is due to low rectal compliance. Irritable bowel syndrome, radiation proctitis, rectal resection and inflammatory bowel diseases are common causes of decreased rectal compliance. Continence to formed stools but incontinence to liquid stools may be caused due to sphincter injury. Anal surgeries unmask subtle abnormalities in continence and sphincter function. Thus this is extremely important to identify these

abnormalities and document them before taking up a patient for anal surgery.

All individuals have primary haemorrhoids. Some mucosal vascular cushions are seen at classical hemorrhoidal sites: 3, 7 and 11 o'clock position even in cross sectional studies of fetal anus. This position correspond to normal venous drainage and arterial supply of anal canal by superior hemorrhoidal artery. Located in the left lateral, right anterior and right posterior part of the anal canal are highly specialized vascular structures forming discrete masses of thick submucosa containing blood vessels, elastic and connective tissue. These act like cushions and also aid in maintaining the anal continence. They are called hemorrhoids only when they become abnormally dilated causing trouble to the patients. Hemorrhoidal veins do not have valves in contrast to the varicose veins which becomes dilated due to valvular incompetence. Hemaorrhoidal veins tend to get engorged following any increase in intra-abdominal pressure due to space occupying lesion or while performing Valsalva maneuver due to decrease in the venous return.

A hemorrhoidal mass is not a varicose vein situated at the termination of the portal venous system.

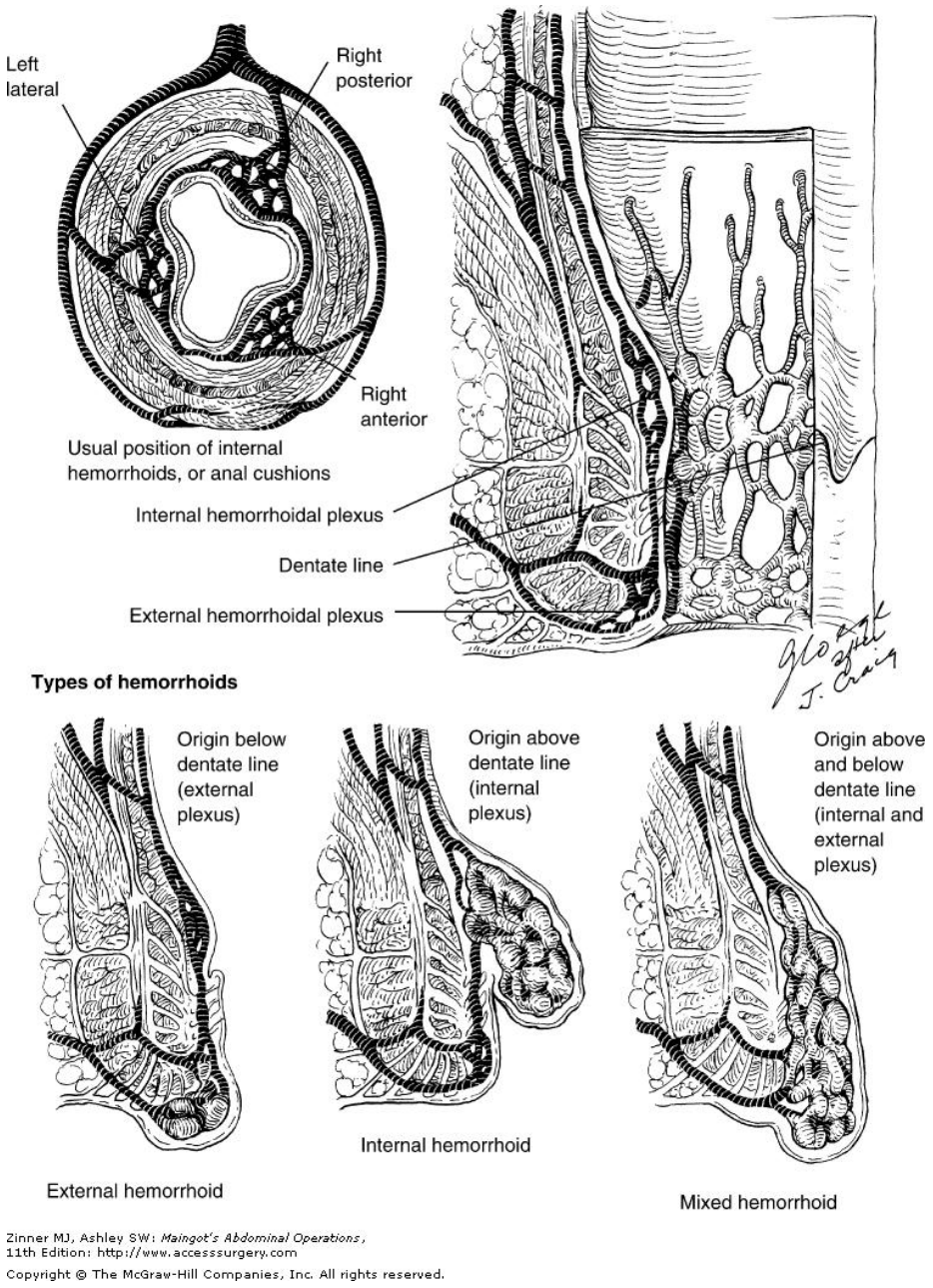
Chronic straining causes disruption of smooth muscle and connective tissue followed by excessive engorgement of the vascular cushions. This disruption causes the anal vascular cushions and the overlying mucosa to prolapse and slide down the anal canal manifesting as hemorrhoids. Repetitive stretching causes further engorgement of veins and promotes prolapse. Irregular bowel habits, gravity, straining, occupation, lack of fibre in the diet, amount of oral intake of water, caffeine consumption are found to have a role in the development of hemorrhoids, the cause of which is unknown and is still being under study.

A clear understanding of the pathophysiology is important when considering therapeutic interventions. At the earlier stages of disease progression, when the major manifestation is transudation of blood through thin-walled, damaged veins and/or arterioles, ablation of the vessels should be adequate. Conversely, in late stages of the disease, when there is significant disruption of the mucosal suspensory ligament, a technique requires fixation of the mucosa to the underlying muscular wall for effective therapy. Internal anal sphincter dysfunction may play a role, since a number of investigators have demonstrated increased internal anal sphincter tone in patients with hemorrhoidal disease. In

reality, probably a combination of all of these factors is important for the ultimate development of large prolapsing hemorrhoidal disease.

Hemorrhoids can be internal, external or mixed based on the dilatation of the anal cushions above or below the dentate line. It can be diagnosed based on history of presenting illness, physical and per rectal examination and endoscopy which can be a proctoscope or a sigmoidoscope.

Fig – 8 Location and types of hemorrhoids.



External hemorrhoids are seen in the anal verge distal to the dentate line and they present as painful defaecation, whereas internal hemorrhoids arises proximal to the dentate line and they present as bleeding, prolapse of mucosa and are usually painless unless they are

thrombosed. The bleeding may be squirting or dripping in the toilet bowl, vary in quantity and frequency and is bright red in colour mixed with stool. Massive bleeding is rare and can occur in cases of excessive straining or with coexistent portal hypertension, where an alternative diagnosis of anorectal AV malformations, bleeding polyps and rectal varices should be considered. Massive bleeding is treated with blood transfusion and urgent hemorrhoidectomy.

CLASSIFICATION OF HEMORRHOIDS

GRADE I	ONLY BLEEDING, NO PROLAPSE
GRADE II	PROLAPSE WITH SPONTANEOUS REDUCTION, BLEEDING AND SEEPAGE
GRADE III	PROLAPSE REQUIRING DIGITAL REDUCTION, BLEEDING AND SEEPAGE
GRADE IV	PROLAPSED, CANNOT BE REDUCED STRANGULATED

Internal hemorrhoids can be classified into various degrees according to their extent of prolapse and the treatment options are stratified based on their degree of presentation. Hemorrhoids that prolapse down the anal canal during straining are Second degree internal haemorrhoids. Prolapsed haemorrhoids requiring manual reduction are Third degree internal haemorrhoids. Masses that cannot be reduced are Fourth degree internal haemorrhoids. Prolapse is more evident during pregnancy and puerperium or if the patient is constipated. Although this staging system tends to correlate with patient's symptoms, it is unclear that it can be completely relied on when making therapeutic decisions. As outlined later, it is important to consider the relative role of internal hemorrhoidal tissue as well as external hemorrhoidal skin tagging when choosing a modality for complete resolution of all of the patient's symptoms.

Mixed hemorrhoids are those in which both internal and external components are present. The mucosal component of mixed hemorrhoids occasionally can be treated by elastic ligation. Large symptomatic, nonreducing mixed hemorrhoids generally are treated by excisional hemorrhoidectomy.

Primary hemorrhoids can be subclassified as:

- Due to reduced anal canal tone
- Due to anal hypertension

Men younger than 40 years of age presenting with first degree hemorrhoids have high anal pressures, whereas women older than 40 years presenting with prolapsing hemorrhoids have reduced resting anal canal pressure.

Secondary hemorrhoids are seen in:

- Chronic obstructive airway disease
- Cirrhotic patients with ascites
- Chronic strain from urethral stricture or prostatism
- Intra-abdominal space occupying lesion like ovarian tumor
- Large rectosigmoid carcinoma
- Distended bladder
- Decompensated liver disease patients with portal hypertension
- Excessive fibrosis of rectal mucosa after radiation therapy due to impaired venous return.

Anemia is usually rare and if present needs further evaluation. Mucosal prolapse can cause mucus or fecal discharge associated.

Irritation of perianal skin produces burning and itching sensation (pruiritis ani). It is not a symptom of external haemorrhoids as it is independent of redundant skin or underlying venous plexuses. Prolapsed internal hemorrhoids increases peri-anal moisture and may cause itching. Presence of pain associated with internal hemorrhoids which is not thrombosed, should lead to evaluation of other causes like fissure and abscess. Thrombosed external hemorrhoids appear as bluish skin covered masses on the anal verge, are firm and painful, causing maximum discomfort for first 48 – 72hrs, with residual discomfort for a week leaving a lump which may take several weeks to resolve. Excision of thrombosed hemorrhoids within 72hrs of occurrence relieves more pain than it causes. The differential diagnosis of thrombosed haemorrhoids is external plexus hematoma which is a self limiting condition resolving within 5 days. It presents with swelling and severe anal pain, and is treated conservatively. For patients presenting with excruciating pain from localized hematoma, rapid resolution is obtained by evacuation of hematoma.

True thrombosed external hemorrhoids is self resolving over 10 – 14 days. If internal hemorrhoids are not reduced and is acutely prolapsed, they may cause engorgement and edema of ipsilateral external hemorrhoidal complex.

Examination of the patient during straining, digital rectal examination and proctoscopic examination is mandatory prior to the confirmation of the diagnosis. It is preferable to examine the patient in the left lateral position with the knees drawn up toward the chest as high as possible. This approach allows relative patient comfort and the ability to clearly inspect the perianal skin and perform anoscopy and proctosigmoidoscopy. A careful digital examination of the anal canal and distal rectum should be performed to include the prostate in men. An anoscope is essential to clearly inspect the hemorrhoidal tissue and anal canal. The three common locations for hemorrhoids should be inspected, and the size, friability, and ease of prolapse of these areas should be recorded. Following this, the decision regarding the need for more proximal colorectal evaluation should be considered, although rigid proctoscopy would be the minimum in all patients. After the hemorrhoids are appropriately graded, a discussion can be enjoined with the patient regarding treatment options.

On inspection of the anal verge after asking the patient to squat and strain, hemorrhoids may appear as two or three circumscribed swellings, in contrast to full thickness rectal prolapse which appears circumferentially. Moreover rectal prolapse patients have a patulous anus which is absent in patients with hemorrhoids. Also there is a

chance of squamous metaplasia over thickened columnar epithelium of large haemorrhoids. Digital rectal examination helps us to rule out other cause of bleeding per rectum like anal neoplasm, cutaneous malignancy of the anal verge and also to assess the resting tone of the sphincter. Patients presenting with severe pallor, age of the patient greater than 40yrs, family history of colonic malignancy and the degree of hemorrhoids not correlating with severity of anemia are subjected to colonoscopic examination, contrast radiology and barium enema studies. Treatment options can be non surgical or surgical based on the degree of hemorrhoids.

TREATMENT:

The principle of treatment is not to improve the appearance of anal canal rather it is focussed towards alleviation of symptoms. Treatment is not justifiable in asymptomatic patients as the operative approach can cause more harm than the underlying pathology. Patients with intermittent, minor symptoms are taught to defecate only after an urge. They should refrain from straining and to increase the volume of the stool, bulk laxatives are prescribed. 85% of the patients presenting with haemorrhoids can be managed by outpatient treatment. But there is always a 20% – 25% chance of recurrence rate within a period of 5 years in patients undergoing outpatient treatment, which always does

not cure the disease. Therefore they must be advised to review periodically. Nowadays Maximum Anal Dilatation (MAD) is no longer advised for patients presenting with tight anal canal and hemorrhoids for fear of iatrogenic anal incontinence.

For outpatient treatment, preoperative preparation is not necessary. Based on the treatment approach postoperative management and follow up is advised appropriately, to the patients undergoing outpatient treatment. These patients can be discharged after the procedure and advised to review if they develop recurrence, for further evaluation and repetition of the same or alternative procedure if necessary. For patients undergoing hemorrhoidectomy, they are warned of complications like reactionary or secondary hemorrhage, anal incontinence or stenosis of the anal canal. They are usually reviewed after a week to ensure that these patients are free of complications.

Non operatively symptoms of patients can be relieved by simple measures like avoidance of excessive straining, better local hygiene like patting the anal cushions than rubbing it after defecation, dietary fibre supplements and plenty of oral intake of water and by medications which help in keeping the stool formed soft and regular. Increasing the fibre content of the diet to at least 25–35 grams daily with raw vegetables, fruits, whole grain cereals, and hydrophilic bulk-forming

agents can reduce and often alleviate all symptoms. Syrup liquid paraffin and tablet dulcolax at night, topical application of local anaesthetic or a steroid-based suppository for pain or use of glycerine suppositories for incomplete rectal evacuation, are the most often prescribed drugs.

Patients with complaints of only bleeding without prolapse of rectal mucosa can be treated dietary fibre supplementation alone for thirty to forty five days. Second and third degree internal haemorrhoids without internal haemorrhoids can be treated with office procedures like injection sclerotherapy, bipolar electrocoagulation, laser or infra red coagulation or a heater probe to produce mucosal fixation. Outpatient treatment is based on the principle of fixing the mucosa above the prolapsing hemorrhoids and is suitable for first degree, second degree and some third degree hemorrhoids.

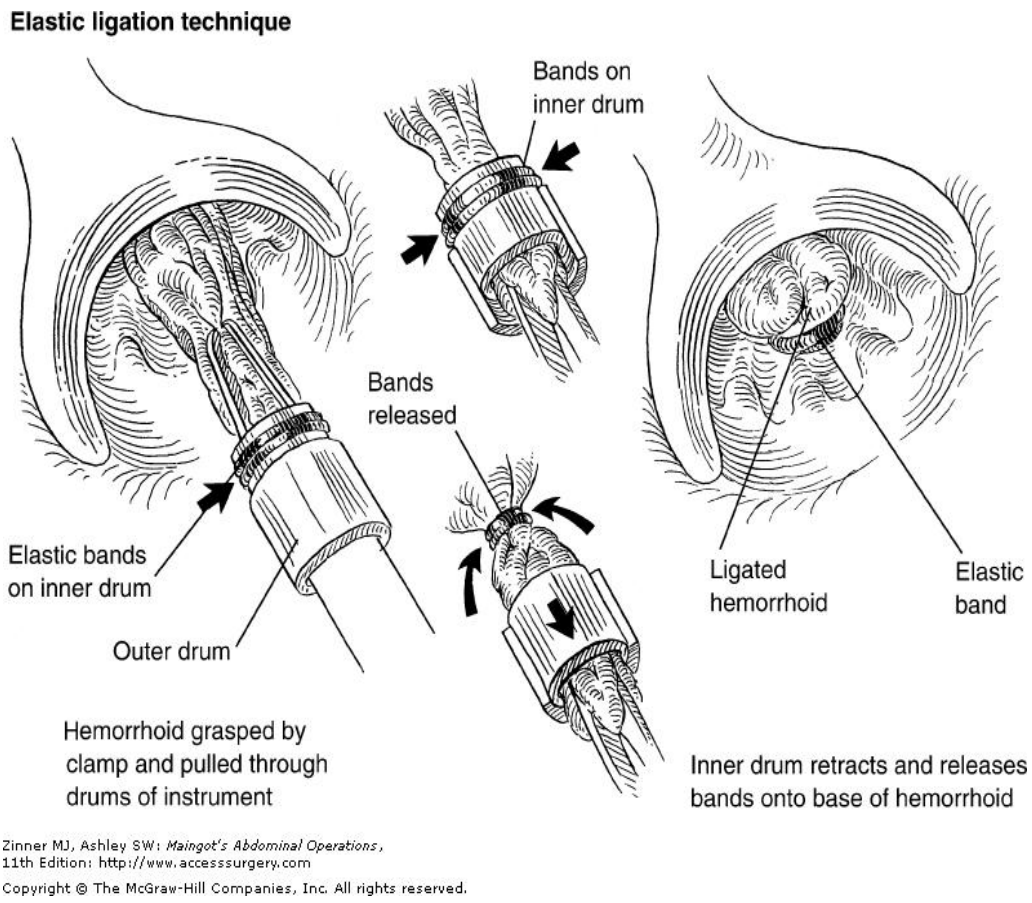


Fig – 9 Elastic ligation technique.

The principle of rubber-band ligation is to apply a rubber band to the mucosa above each hemorrhoid without disturbing the pile mass. Suction-band technique is preferable as it does not require the help of an assisting surgeon. Usually only one hemorrhoid is ligated on the first treatment visit. Ligations can be performed every 2–4 weeks until all symptoms of bleeding or prolapse are alleviated. The second ligation can be multiple if the first treatment is well tolerated**.** Rubber band is applied after sucking the mucosa into the cup of applicator thereby leaving rectal mucosa like a polyp, with the base of the polyp snared by

the rubber band. The ligatures must be placed at least 1–2 cm above the dentate line to avoid extreme discomfort. Ideally, the ligatures should be placed at the top of the hemorrhoidal cushion. Band causes ischemic necrosis of hemorrhoid followed by the development of an ulcer, which heals by fibrosis thereby preventing the slipping down of hemorrhoids. About 25% of patients experience mild, dull anorectal discomfort lasting for 2–3 days following the procedure. Mild analgesics and warm baths are usually sufficient to relieve the discomfort. After first application 70% of patients are rendered asymptomatic and this increases to 90% after two or three applications. About 2% of patients treated with ligation of the internal hemorrhoid develop thrombosis of an external hemorrhoid, which may cause considerable discomfort. Of all the procedures available rubber band ligation is most effective and most widely applied office procedure without sedation but can be complicated by post operative pain, tenesmus, transmural ischemic necrosis, infection, sepsis and death in few occasions. This also requires the appropriate instrument (fenestrated anoscope e.g., Hinkel-James type as well as the McGivney rubber band applicator) which is not easily available everywhere; and the technical skill of the surgeon in performing the procedure.

Sclerotherapy of symptomatic internal hemorrhoidal disease was first advocated by Mitchell in 1871 and has enjoyed significant experience. The principle of injection sclerotherapy is to place 2ml – 5ml of sclerosant just above the hemorrhoid in the submucosal plexus. Sclerosant is injected into the mucosa, above each of the principal hemorrhoid. It can cause hematuria, prostatitis and hematospermia if accidently injected deeply into the bladder, prostate or seminal vesicle. This is important while injecting sclerosant anteriorly and extreme caution is to be exercised. The amount of sclerosant injected under the mucosa must be sufficient enough to raise a white wheel at the site of injection. This causes ulceration, fibrosis and finally fixation of mucosa preventing its further descent into the anal canal. Complications are pain, bleeding, hematospermia, urethritis, hematuris and retention of urine which is seen in less than 2% of patients. Further injections are not required in 90% of cases. Injection sclerotherapy though promising equal results, in comparison to rubber-band ligation is relatively easier, cheaper and is associated with less post operative complications. Many clinicians find injection sclerotherapy more satisfactory, which is invasive and difficult to administer in a busy setting clinic. Moreover high risk patients undergoing rubber band ligation are to be put on prophylaxis for subacute bacterial endocarditis and they are not suitable for patients taking antiplatelets or other blood-thinning medications. In

immunodeficient patients rubber band ligation is to be avoided. Patients undergoing rubber-band ligation should be warned that there is a 5% chance of delayed bleeding (with anal discomfort within 48 hours in these patients) between 7^{th} and 10^{th} postoperative day due to separation of slough; which is absent after sclerotherapy.

Over the past 10 to 15 years, a variety of new devices have been advocated for hemorrhoidectomy. These energy-based cutting devices have been devised to allow simultaneous tissue division and coagulation. The main advantage proposed for these devices is provision of hemostasis without need for suture ligation and therefore reduction in postoperative pain. However, these benefits must be interpreted in the context of the significant cost of acquisition of the devices compared to a disposable scalpel blade.

Mucous discharge is a common complication after cryotherapy and therefore it has been discontinued.

Photocoagulation is reserved first degree haemorrhoids as it is less effective than rubber band ligation. Photocoagulator probe is applied about three or four times to the mucosa above each hemorrhoid creating a white area of burn at the end of this procedure. Though postoperative complications are minimal, it is more painful than above mentioned procedures. No further treatment is required in 70% of cases.

The first energy cutting tool applied to hemorrhoidectomy is standard monopolar electrocautery. The tool has been reported widely for the two dominant types of hemorrhoidectomy. Surgeons using this tool have also employed various degrees of wound closure by suture, ranging from pedicle ligation only to complete wound closure. Bipolar diathermy employs electrical current to coagulate the hemorrhoidal tissue, including the mucosa and submucosa. The machine generates a 2-second pulse of energy to accomplish the treatment. Once again, this approach is applicable for small bleeding hemorrhoids and probably has no greater efficacy than does sclerosing.

Other variations on the use of energy to destroy internal hemorrhoids includes infrared coagulation and Ultroid (direct-current) therapy. The infrared coagulation employs a tungsten halogen lamp that generates heat energy generally for a 1.5-second period resulting in destruction of the mucosa and submucosa at the application site. The depth of penetration of this injury is usually 3 mm. Conversely the Ultroid uses electrical current that is applied for up to 10 minutes per complex treated. Ultimately, all of these new modalities are a variation on the theme of local tissue destruction and fixation of the hemorrhoidal tissue at the appropriate level. There is probably no advantage of one

technique over the other; however, sclerotherapy offers an advantage to the physician since minimal instrumentation is required.

Using cautery or laser, limited hemorrhoidectomy can be performed intra-anally. From the internal sphincter hemorrhoidal tissue is dissected and hemostasis is achieved by laser, or by spray or contact cautery. Either Nd-YAG or CO_2laser excision may be performed; however, it is not clear that the added expense or benefits are superior to scalpel or scissor excision. It can be done on the physician's office or as an outpatient treatment and is appropriate for small second degree hemorrhoids. Reactionary or secondary hemorrhage is most common complication of this procedure.

Ligasure or suture ligation of hemorrhoids is also appropriate for small second degree hemorrhoids and involves ligating the hemorrhoidal pedicle using a specific applicator. A bipolar cautery device capable of simultaneous tissue division and blood vessel coagulation is the LigaSure. This device has been compared to monopolar diathermy hemorrhoidectomy with most of the data suggesting reductions in operative time and early postoperative pain. It often recurs.

Surgical treatment: Hemorrhoidectomy is the gold standard of all the treatment options available and is done for patients presenting with

grade three and four haemorrhoids and for those who have failed by non operative measures. Cold scalpel or scissor excision has long been the mainstay of surgical hemorrhoidectomy, and the data on outcomes are well validated. Patients should always be counselled preoperatively that the procedure is painful and is also associated with early complications like bleeding and sepsis, and delayed complications like anal stenosis and impairment of anal continence. Rare complications include:

- Postoperative fissure
- Unhealed open wounds
- Submucous abscess

In controlling prolapse and bleeding hemorrhoidectomy is extremely successful in majority of the cases.

Excision is sufficient for simple thrombosed external hemorrhoids. Patients with severe grade four haemorrhoids with surrounding edema are initially treated with magnesium sulphate salt and glycerine dressing + sitz bath to minimize the edema. These patients usually have anal tags, which is a skin overlying the prolapsed part of the hemorrhoids. They are subjected to hemorrhoidectomy later, once the edema gets subsided. Treatment options for these patients are dictated based on the time of presentation. If the time duration is greater than 72hrs then conservative line of approach with analgesics and stool

softeners may be appropriate. If the presentation is less than 72hrs then hemorrhoidectomy is reasonable. Patients with grade four haemorrhoids presenting with bleeding are subjected to urgent hemorrhoidectomy in some centres. Conventional hemorrhoidectomy can be open or closed and can be performed as an outpatient procedure. In open procedure any discharge like pus, serum or blood can be drained easily but there is a skin defect. Whereas in closed procedure the defect is closed leaving an intact anal canal. The sphincteric function and postoperative pain following each of these procedure is similar. Thus the choice of procedure is based on individual's preference.

The choice of anaesthesia depends on the preference of the patient, surgeon or the anaesthetist based on the co-morbid factors and the general build and nourishment of the patient.

Complex internal and external haemorrhoids can be excised by closed hemorrhoidectomy. Patient is usually operated in prone Jackknife position, hip flexed to 40 degrees with buttocks strapped apart. The use of large operative Fansler's scope plays an important role. The best way to identify the hemorrhoid is to place a gauze in the anal canal during proctoscopic examination. Scope is removed and the gauze placed inside the anal canal is slowly withdrawn which ultimately exposes the dilated vein and helps in its identification. Under general anaesthesia or

sub-arachnoid block infiltration of local anesthetic + weak adrenaline of 1:200,000 dilutions is given into the subcutaneous and submucosal planes of each hemorrhoid to minimize bleeding during operation. This also helps in easy separation of pile mass from internal sphincter. With tissue forceps the anal skin tag is lifted up and is cut away from internal sphincter using heel of scissors. The dilated hemorrhoidal vein is identified and dissected on either side of the defect from beneath the cut edge of mucosa. This helps in formation of two mucosal flaps which is closed at the end starting from the apex, which is anchored to the internal sphincter. Absorbable suture on a cutting needle is used for closure of the defect. Subcuticular technique is used to approximate the skin of anal verge and to prevent placing of knots at the apex.

Complete hemostasis is achieved by this procedure which is confirmed by inserting a Pratt's operating scope at the end of the procedure. Meticulous hemostasis is extremely important in this procedure since the injected adrenaline may mask bleeding by causing vasoconstriction and the bleeding may also not be more evident in Jackknife position as it may gravitate into the anal canal. Achieving hemostasis from each vessel as it is transacted is difficult as it is a vascular complex with multiple channels fed by many small vessels. Goldberg in 1980 pointed out that much of the bleeding comes from

mucosal incision and hemostasis can be conveniently attained by application of coagulating electrocautery.

Sufficient bridge of skin in the anoderm can be preserved thereby preventing anal stenosis; which is a long term complication following fibrosis of the anal verge if sufficient bridge of anoderm is absent. Urinary retention, bleeding, fecal impaction and infection are the usual post operative complications associated with surgical management of haemorrhoids. Patients usually recover in one or two weeks. Postoperative fever is to be immediately reported as closed procedure is often complicated by submucosal and intersphincteric abscesses.

Alternatively hemorrhoidectomy can be left open than closing them primarily, but the healing rate is longer. Open technique may be performed in lithotomy or prone Jackknife position. After anesthesia local infiltration is given into submucosal plane of each hemorrhoid and into the lower part of intersphincteric space. It is preferable to start with pile mass in 7 o'clock position, proceeding to 3 o'clock and finally the 11 o'clock so that bleeding does not obscure the operating field.

Skin adjacent to pile mass is held using tissue forceps and an inverted V-shaped incision is made in the perianal skin after grasping the tissue forceps using left hand. The dissection is deepened upto the level of internal anal sphincter. The base of hemorrhoidal pedicle is

identified where the arterial supply and venous drainage is present. The internal sphincter is pushed away and the base of pedicle is transfixed and doubly ligated thereby completing hemorrhoidectomy. After ensuring hemostasis anal pack is kept in situ and T – bandage is applied. The advantage of leaving it open helps in reducing the post operative pain.

Complications of conventional hemorrhoidectomy include:

- Due to restoration of normal blood pressure or from slippage of poorly secured knot, reactionary hemorrhage can occur in first 24 hours. Emergency exploration may be necessary.
- Between 5^{th} to 8^{th} postoperative day, secondary hemorrhage can occur necessitating emergency operation due to erosion of vessel from sepsis. It can be treated by ligation and antibiotics.
- Elderly patients are often complicated by acute urinary retention which may require catheterization.
- Postoperative pain may be severe and is managed by analgesics like topical or oral NSAIDs. Avoid opiates. As patients are dehydrated and immobilized laxatives and stool softeners are essential.
- Fecal impaction is yet another complication and may aggravate the pain.

- Unhealed closed or open wounds are treated with topical diltiazem or nitrates.
- Soiling and flatus incontinence may occur if internal sphincter is injured.
- When the skin bridge is inadequate the retained skin may undergo ischemic necrosis, if the base is not wide enough and eventually may lead to the development of anal stenosis (Whitehead deformity). This condition is difficult to be managed and requires anal dilatation leading to restenosis and further anal stretching, thereby causing damage to the residual sphincter. Bilateral rotating anoplasty or anal skin advancement procedures may be beneficial. Restenosis and sepsis occurs in 20% - 30% of anoplasty cases.

Recent advances in the management of hemorrhoids include the application of ultrasonic or controlled electrical energy like Harmonic Scalpel and LigaSure for excision of hemorrhoids.

A competing technology is the Harmonic scalpel, which relies on a rapidly reciprocating blade to generate heat for coagulation and tissue transection. The overall postoperative complication rates were low, with urinary retention in 2%, fissure in 1%, and abscess/fistula in 0.8%. The postoperative hemorrhage rate is as low as 0.6%. The post operative

pain associated with these procedures are very minimal. They help in sealing the dilated vessels and removal of prolapsing mucosal tissue. Moreover thermal injury to adjacent lateral structures is minimal with these procedures thereby reducing the post operative pain and edema.

Another newer technique devised by Longo is the usage of circumferential stapled hemorrhoidectomy or stapled hemorrhoidopexy where circumferential excision of mucosa and submucosa of rectum and anal canal is done, followed by reanastomosis and fixation in their normal anatomic location using a circular stapling device. This approach can be done using a dedicated device which has an obturator and a circular stapler. The proctoscope remains fixed till the completion of surgery. A 31-mm stapler is then placed transanally to perform a circumferential excision of rectal mucosa just rostral to the hemorrhoidal columns. The prolapsing mucosa should be repositioned prior performing this procedure.

About 3 to 4cm above the dentate line a circular purse-string suture is placed which should include all the redundant mucosal tissue; with each suture taken at submucosa level, rather than taking it full thickness, to avoid complications associated with it. If the suture is taken too much proximally without incorporation of mucosa, the symptoms would not get relieved and if the suture is taken at the level of

dentate line, urgency and severe pain may complicate. After purse string sutures stapling gun is introduced after which one cannot be sure of whats happening inside anal canal. After removal of circular stapler, the excised ring of tissue should be inspected and sent for HPE (histology) to ensure the absence of muscle entrapment. Rectal integrity, the staple line and rectovaginal septum (in female) is assessed by proctosigmoidoscopy and bimanual palpation at the end of the procedure.

As a result of excision of anal mucosal cylinder skin tags may improve but it has no influence over skin tags directly. This procedure is best in providing short term benefits but most often recur. This overnight stay procedure is painless in 80% - 90% of cases with good long term results. Though painless, it is not safe to advise a patient to undergo this procedure without explaining the risk of complications associated with this procedure.

Complications of this procedure are:

- rectal perforation,
- severe pelvic sepsis,
- anastomotic dehiscence,
- rectovaginal fistulas
- pain (anterior resection syndrome)

- chronic symptoms of tenesmus
- incomplete rectal evacuation

The reason for complications associated with this procedure is that this procedure is blind, and it is not possible to check the correctable problems during excision process after firing the gun. These complications which were common after stapler hemorrhoidopexy have become minimal nowadays due to improvement in technical skill of surgeons.

Hemorrhoidal Artery Ligation (HAL) under ultrasound guidance using trans-rectal probe is being tried nowadays. The outer sheath of the transparent self illuminating proctoscope is provided with a window through which the ligature is taken after tracing out the arterial pulsation. The suture is taken continuous towards the anal verge and finally knotted so that the prolapsing anal mucosa is repositioned into its normal anatomic location by tightening of the sutures. The long term results of this new technique are yet to be established.

POSTOPERATIVE CARE:

Regardless of the excisional technique used for treatment of advanced hemorrhoidal disease, the key to effective patient management is avoidance of postoperative complications.

Pain is the most frequent complication and is the most feared sequelae of the procedure from the patient's perspective. A variety of analgesic regimens have been recommended, usually consisting of a combination of oral and parenteral narcotics. The use of local infiltration of bupivacaine into the wounds and perianal skin has been variably successful in long-term pain reduction. Conversely, ketorolac has demonstrated considerable efficacy in managing posthemorrhoidectomy pain. The use of alternative administration routes for narcotics either by patch or subcutaneous pump have been successful in controlling pain; however, the management of these routes of administration can be risky in the outpatient setting because of the risk of narcotic-induced respiratory depression. The most appropriate regimen following outpatient hemorrhoidectomy appears to be intraoperative use of ketorolac, sufficient doses of oral narcotic analgesics for home administration, and supplementation of the narcotics by an oral nonsteroidal medication.

Urinary retention is a frequent postoperative problem following hemorrhoidectomy, ranging in incidence from 1% to 52%. A variety of strategies have been used to treat the problem, including parasympathomimetics, α-adrenergic blocking agents, and sitz baths. The best approach, however, seems to be a strategy of prevention that

includes limiting perioperative fluid administration to 250ml, an anesthetic approach that avoids use of spinal anesthesia, avoidance of anal packing, and an aggressive oral analgesic regimen.

Early **postoperative bleeding** (<24 hours) occurs in approximately 1% of cases and represents a technical error requiring return to the operating theater for resuturing of the offending wound. Delayed hemorrhage occurs in 0.5% to 4% of cases of excisional hemorrhoidectomy at 5 to 10 days postoperatively. The etiology has been held to be early separation of the ligated pedicle before adequate thrombosis in the feeding artery can occur. The bleeding in this scenario is usually significant and requires some method for control of ongoing hemorrhage. Options include return to the operating theatre for suture ligation or tamponade at the bedside by Foley catheter or anal packing. The subsequent outcome after control of secondary hemorrhage is generally good with virtually no risk of recurrent bleeding. It may be helpful to irrigate out the distal colorectum with posthemorrhage enemas or at the time of intraoperative control of bleeding to avoid confusion when the residual clots pass per anum.

MATERIALS AND METHODS

All patients who presented with grade I and II hemorrhoids to the Government Stanley Medical College Hospital were included in the study. The other causes of bleeding PR like fistula and fissure in ano, inflammatory bowel disease and rectal malignancy were excluded from the study. These patients were treated on outpatient basis fixing up a day called the sclerotherapy day, every week. All the patients were free of comorbid disease like diabetes, hypertension, tuberculosis and cirrhosis. A small set of additional patients with pre-existing comorbid factors were also subjected to sclerotherapy. A total of 100 patients were studied excluding the small set of patients.

The purpose of sclerotherapy is ultimately to scar the submucosa, resulting in atrophy of the tissue injected and scarification with fixation of the hemorrhoidal complex within its normal location in the anal canal. A variety of solutions have been advocated, although it appears that sodium morrhuate and sodium tetradecyl sulfate predominate currently. This modality is most effective in situations with minimal enlargement of hemorrhoidal complexes where the primary complaint is bright red rectal bleeding.

The procedure is performed with the patient in the left lateral decubitus position. An self illuminating anoscope or a proctoscope which is illuminated by an external source of light, is inserted to clearly identify the symptomatic complex and a 25-gauge spinal needle is used to instill the sclerosant into the submucosal space (Fig.). Gabriel's syringe, was used in olden days to instill the sclerosant. The syringe should be aspirated prior to injection to avoid a direct intravascular injection. Typically 1 to 2ml of sclerosant is injected submucosally sufficient enough to raise a wheel at the injection site. Alternatively 1ml of sclerosant diluted with 2ml of distilled water and reconstituted to 3ml can be injected into each hemorrhoid. The injections are to be directed to the base of hemorrhoid (pedicle). Intravenous injections are to be avoided as the absence of valves in these veins can cause systemic entry and adverse reactions. The surgeon can inject as many locations as desired because the procedure is essentially painless. It is important, however, not to circumferentially inject the anal canal because this may induce stricture formation.

Local discomfort following injection can be minimized by prior infiltration of local anesthetic which may not be often necessary.

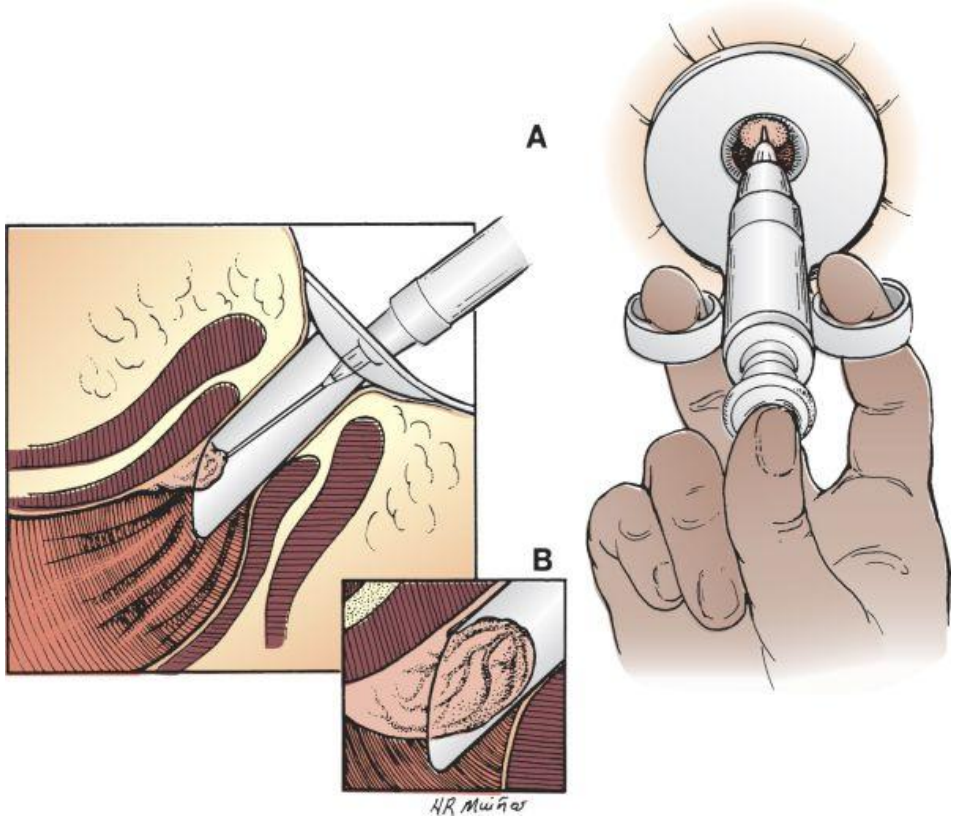


Fig - 10 **A,** Injection of internal hemorrhoid. **B,** Postinjection striations.

Immediately after sclerothereapy few patients experience anal discomfort, hence one ml of sodium tetradecyl sulphate mixed with 2 ml of 2% lignocaine is reconstituted to a total of 3 ml by few surgeons and 1ml of reconstituted solution is injected into bash of each hemorrhoid. No drug interaction is noted by mixing the sclerosant with local anesthetic in the same syringe.

OBSERVATION AND RESULTS

After injection sclerotherapy dilated hemorrhoidal vein collapses and shrinks within a week. The inflammatory reaction initiated by sclerosants causes ischemic necrosis of the dilated vein making it to disappear in a week time. The collapsed vein following fibrosis is naturally absorbed by the body and eventually disappears over a period of time. It takes only ten minutes to perform this non-invasive procedure and is more appropriate for grade I and II hemorrhoids. It is curative for grade I and few grade II hemorrhoids, but treatment results for grade II masses is not promising and tend to recur within a year. For pile masses of grade III and IV, the size of mass is found to decrease following this procedure which is not consistent. These higher grades, does not completely disappear and most often require repeated injections for better control of symptoms.

MERITS:

- all the three hemorrhoids can be simultaneously treated,
- it is comparatively cheaper, easier and can be performed in doctor's office as an outpatient procedure.

- Patients with weekend vascular structures can also be treated and the results persist for a period of one year.

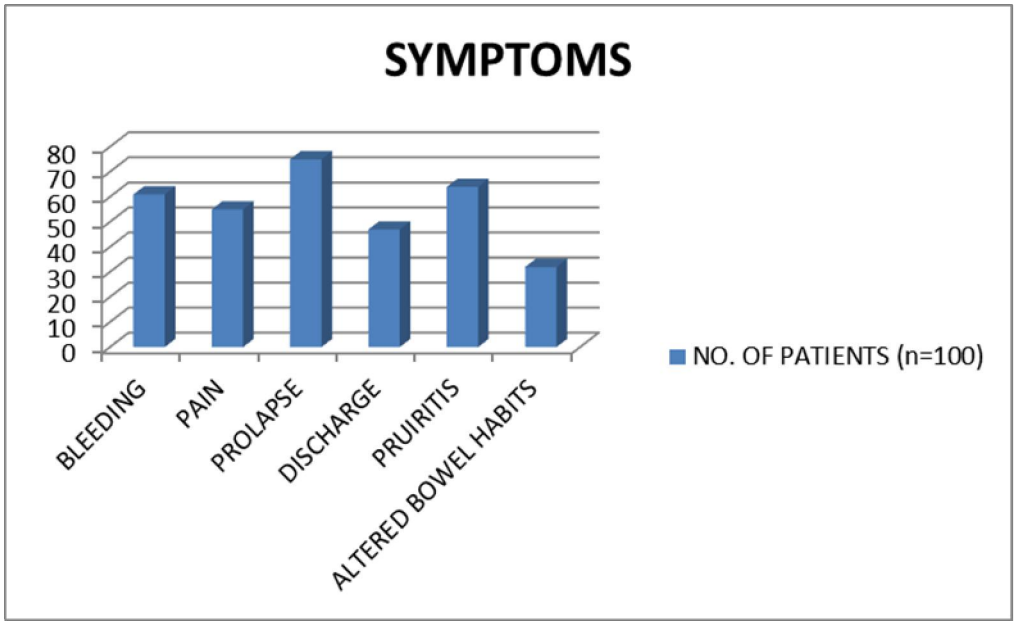
DEMERITS:

- Hemorrhoids of larger size have high failure rates,
- The recurrence is almost certain within one year period.

RESULTS:

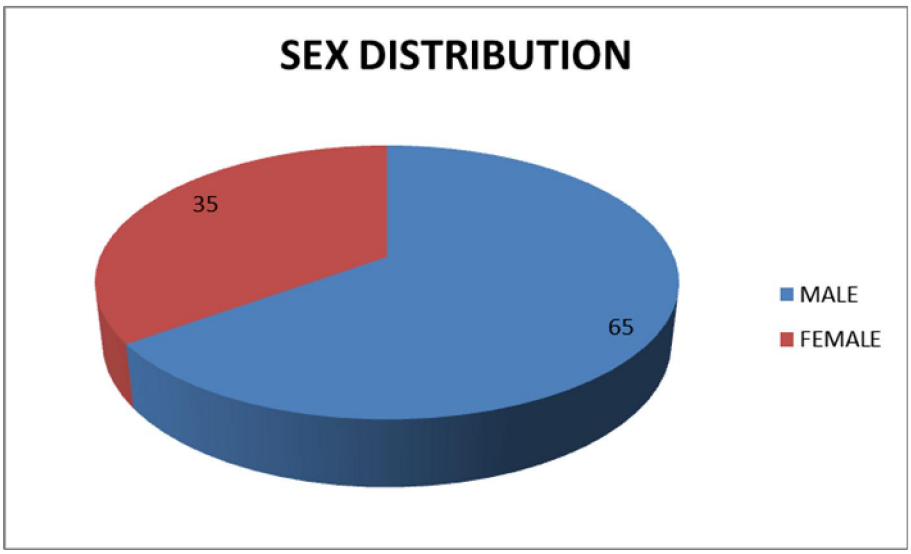
Total number of patients presenting with symptoms of hemorrhoids were:

SYMPTOMS	NO. OF PATIENTS (n=100)
BLEEDING	61
PAIN	55
PROLAPSE	75
DISCHARGE	47
PRUIRITIS	64
ALTERED BOWEL HABITS	32



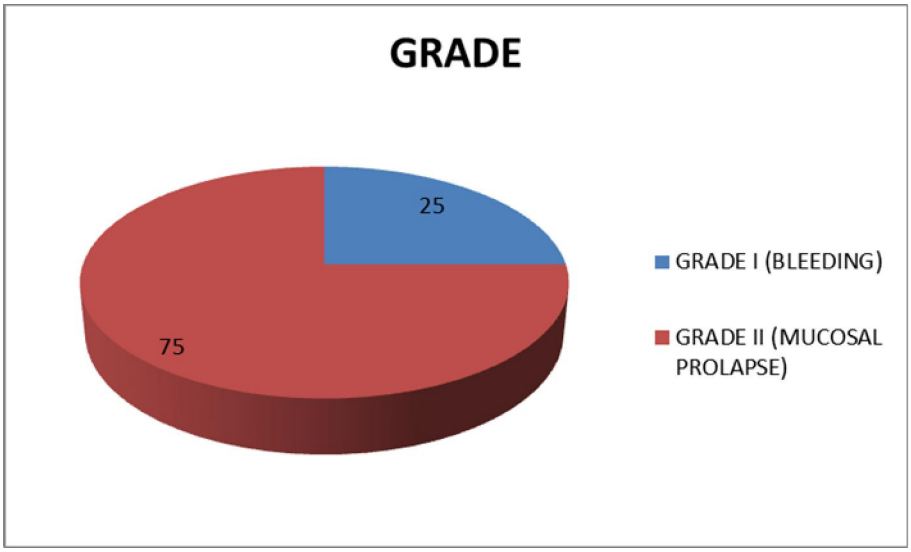
Total number of males were 65 and females were 35 in a ratio of 2:1 approximately, who presented with symptoms to the general surgical OPD.

GENDER	NUMBER OF PATIENTS
MALE	65
FEMALE	35



Of those patients, 61 patients had bleeding and 60 of them had prolapse of rectal mucosa.

GRADE OF HEMORRHOIDS	NO. OF PATIENTS
GRADE I (BLEEDING)	25
GRADE II (MUCOSAL PROLAPSE)	75



Following first dose of injection sclerotherapy 7 of them still complained of bleeding and 17 of them complained of mucosal prolapse when they were followed up after one month.

GRADE OF HEMORRHOIDS	NO. OF PATIENTS	PERCENTAGE
GRADE I (BLEEDING)	54	88.52%
GRADE II (PROLAPSE)	43	71.66%

54 of 61 patients with bleeding and 43 of 60 patients with mucosal prolapse had symptomatic relief following first dose.

Thus for bleeding it provides a symptomatic relief of 88.52% and for mass descending per rectum it provides a symptomatic relief of 71.66%.

Patients without symptomatic relief were subjected to second dose of sclerotherapy after one month, the results were:

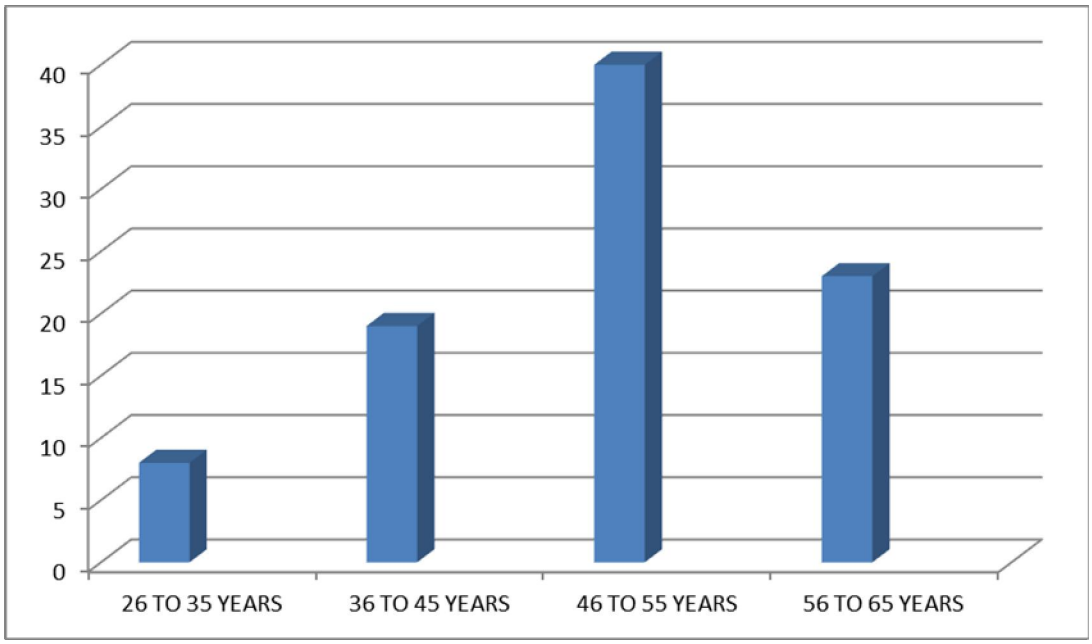
SYMPTOMS	SYMPTOM FREE	PERCENTAGE
BLEEDING	6/7	85.71%
PROLAPSE	12/17	70.58%

The remaining 5 patients of prolapsed were considered as failure of sclerotherapy and were subjected to other modes of treatment.

AGE DISTRIBUTION

The majority of patients were in the age group of 46 to 55 years of age numbering around 40 patients.

AGE GROUPS	NO. OF PATIENTS
26 TO 35 YEARS	8
36 TO 45 YEARS	19
46 TO 55 YEARS	40
56 TO 65 YEARS	23
66 TO 75 YEARS	7
76 TO 85 YEARS	3



MEAN AGE = 51.67 YEARS

Number of patients presenting grade I hemorrhoids were 25.

Of 47 patients presenting with mucoid discharge 35 of them were found to be symptom free.

Therapy did not have much influence on pruiritis as only 22 of 64 stated to be symptom free.

Postoperatively patients complained of constipation as they were prior to sclerotherapy. 4 of 32 patients were symptom free.

Of the 100 patients in the study 6 of them had bleeding following injection sclerotherapy. Other complications like urinary retention, prostatitis, hematospermia and abscess formation were not reported. Portal pyemia, sepsis and death were absent. By the end of one year 5/75 had recurrence even after repeated injections.

10 patients with comorbid factors like anemia,ischemic heart disease were studied, all presenting with grade III hemorrhoids. Single dose of sclerotherapy found to stop bleeding in 8 of 10 patients. The prolapsed hemorrhoid became smaller in size in 7 of 10 patients. These results were short lasting and recur soon. 3 of them had no response to sclerotherapy.

All the patients were discharged on the same day after few hours of observation. Some experienced burning sensation while injecting into the hemorrhoidal pedicle.

DISCUSSION

Sclerotherapy has been proven to be effective in the management of patients presenting with bleeding as the only presenting complaint. In our study, 89% of patients with bleeding were rendered symptom-free after a single dose of sclerotherapy.

72 % of patients with mucosal prolapse showed resolution of their complaints with the first dose itself. Of the remaining 28%, only 7% required haemorrhoidectomy, while the rest were cured of their symptoms by a second dose of sclerotherapy.

Only 35% with pruritus experienced a significant benefit post the procedure, while the majority of patients (75%) complaining of mucoid discharge showed improvement in their symptoms.

80% of patients with other comorbidites with Grade III haemorrhoids experienced resolution of bleeding, but, this group required multiple injections, and the recurrence rate was higher.

These findings are comparable to most international studies.

CONCLUSION

The management of symptomatic hemorrhoidal disease should be directed at the symptom complex of the individual patient. Most of these patients can be successfully treated by improving bowel function, correcting constipation, and using any of a variety of anal ointments. For persistent symptoms, either injection or banding of the internal hemorrhoids is predictably successful. Only a few patients should require excisional hemorrhoidectomy by any of the described techniques. Cir-cular stapled hemorrhoidectomy may prove to be an effective, less painful technique to manage grade III hemorrhoids.

Thus, sclerotherapy is a curative option for Grade I and II hemorrhoids, also serves as a tool for temporary symptomatic control for patients with Grade III haemorrhoids.

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PROFORMA

MERITS AND DEMERITS OF SCLEROTHERAPY IN THE MANAGEMENT OF GRADE I AND II HEMORRHOIDS. A STUDY OF 100 CASES

Patient details **Patient ID No:……**

Name:……

Age/Sex:

IP No:

DOA:

DOS:

DOD:

Address:…….

……. Phone number:……

History

Occupation: **Rural / Urban**

Socioeconomic Status: Upper / Upper middle / Lower middle /Poor

PAINFUL DEFECATION: **Duration:** **Onset:**

BLEEDING PER RECTUM: **Duration:** **Onset:**

ITCHING: **Duration:** **Onset:**

MASS DESCENDING PR: **Duration:** **Onset:**

ALTERED BOWEL HABITS: **Constipation/Diarrhoea**

Family History:

Past History: DM / HT / Asthma / TB / Other (Surgery)

Dietary History:

General Examination:

CONSCIOUS

ORIENTED

AFEBRILE

ICTERIC

ANEMIC

Pulse:

BP:

Per Rectal Examination

Proctoscopic examination

Investigations

Hb:

TC:

DC: P L E M

ESR:

Blood Sugar:

Blood Urea:

Serum Creatinine:

SYMPTOM	SODIUM TETRADECYL SULPHATE
PAINFUL DEFECATION	
BLEEDING PER RECTUM	
ITCHING	
MASS DESCENDING PR	
ALTERED BOWEL HABITS	
MUCOID DISCHARGE	

ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

ஊசி மருந்து சிகிச்சையின் மூலம், மூலத்தினால் ஏற்படும் பிரச்சினைகளை சரிசெய்வதில் உள்ள பயன்கள் மற்றும் கெடுதிகள்

ஆராய்ச்சி நிலையம்

பொது அறுவை சிகிச்சைப்பிரிவு

ஸ்டான்லி மருத்துவக்கல்லூரி, சென்னை – 600 001.

பங்கு பெறுபவரின் எண் .....

பங்கு பெறுபவரின் பெயர் / வயது / விலாசம் .....

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எனக்கு (HEMORRHOIDS) மூலம் இருப்பதை மருத்துவர் மூலம் அறிந்தேன். அதற்கு ஊசி மருந்து சிகிச்சையின் மூலம் (SCLEROTHERAPY) சரிசெய்ய சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் .....

இடம் ..... தேதி .....

ஆய்வாளரின் கையொப்பம் .....

இடம் ..... தேதி .....

MASTER CHART

NAME	AGE	SEX	OP/IP NO	BLEEDING	MASS DESCENDING PR	PAIN	MUCOID DISCHARGE	PRUIRITIS	ALTERED BOWEL HABITS
MUNUSAMY	68	M	91658	+	+	+	-	+	-
KUMAR	49	M	18723	+	+	+	-	-	+
DHIVAKAR	42	M	50892	+	+	-	-	-	+
RAVI	51	M	58314	+	+	+	+	+	-
DINADAYALAN	46	M	18755	-	+	+	+	+	-
SUBRAMANI	53	M	16481	+	+	-	-	+	+
MOORTHY	35	M	44726	+	-	+	+	+	-
SUNDHAR	52	M	17152	-	+	-	-	+	-
KUTIAMMAL	49	F	21104	-	+	+	-	-	-
MANI	55	M	18463	+	+	-	+	+	+
VASANTH	47	M	20141	+	-	-	-	-	-
SURESH	38	M	19487	+	+	+	-	+	-
ABIRAMI	47	F	22547	-	+	-	+	-	-
FATHIMA	29	F	18740	+	-	+	-	+	+
INDHIRA	38	F	17271	+	-	-	+	+	-
VARDHINI	42	F	16003	+	-	-	+	+	-
MALATHI	32	F	15496	+	+	-	-	-	+
INDHUMATHI	39	F	15593	-	+	+	+	-	-
PACHAYAMAL	56	F	15824	-	+	-	+	+	-
AARTHI	41	F	19838	+	-	-	-	+	-
SUSEELA	46	F	65194	+	-	+	-	+	+
BABU	55	M	20196	+	+	-	+	-	-
RAMESH	49	M	19889	-	+	+	+	-	-
SUBASH GANDHI	63	M	18026	-	+	+	-	+	+
LENIN	54	M	15934	-	+	-	+	-	+
PUSHPARAJ	48	M	21060	+	-	-	+	+	-
THIYAGARAJAN	46	M	58422	-	+	+	-	-	-
POOVARASI	54	F	69211	+	+	-	-	+	-
CHITRA	65	F	49880	+	+	+	+	-	+
VALARMATHI	39	F	50298	+	-	+	-	-	-
MAHESHWARI	45	F	61099	-	+	+	-	+	-
SATHISH KUMAR	76	M	18421	+	+	-	+	+	+
RAJ KUMAR	43	M	24167	+	-	+	-	-	+
CHANDRASEKAR	40	M	18094	-	+	-	+	-	-
PUNITHAVADHI	33	F	20162	+	+	+	-	+	-
GOMATHI	53	F	31890	+	+	+	-	+	-
INDRAAMMAL	45	F	20956	-	+	-	+	+	-

SUBHASHINI	62	F	58901	+	+	-	-	+	+
NARASIMHAN	39	M	63092	-	+	-	-	-	-
GIRIDHARAN	61	M	11095	+	+	-	+	+	-
PRABHU	48	M	10293	+	+	+	-	-	-
RAMA MOORTHY	30	M	13065	-	+	+	+	+	+
RESHMI	35	F	52549	+	-	-	-	+	-
SRINIVASAN	47	M	19935	-	+	+	-	+	-
MINU LAKSHMI	53	F	19406	+	+	-	+	-	+
KANDASAMI	32	M	18354	+	-	+	-	+	-
MANIKANDAN	65	M	18048	-	+	-	-	-	-
VIKRAM PRABHU	36	M	17860	-	+	+	+	+	+
JASMINE BANU	46	F	54020	+	+	+	+	+	-
ARUL RAJ	63	M	66937	+	+	-	-	+	+
VARALAKSHMI	54	F	58829	+	+	+	-	-	-
MEENAKSHI	65	F	60383	-	+	+	+	+	-
SURESH BABU	38	M	70939	+	-	-	-	-	-
VENKATESAN	48	M	10757	-	+	+	+	+	-
ARUL MURUGAN	57	M	87749	-	+	-	+	+	-
GANESH BABU	54	M	10284	+	+	-	-	-	+
RAVICHANDRAN	35	M	1749	-	+	+	+	-	+
SELVARAJ	39	M	49497	+	-	+	-	+	-
RAJ KUMAR	65	M	29379	+	+	+	-	+	+
MINNALA	47	F	89655	+	-	-	+	+	-
KALAISELVI	62	F	29839	-	+	-	-	-	+
KEERTHI VARMAN	47	M	87239	+	-	+	+	+	-
ARUN BABU	74	M	38788	-	+	+	-	-	-
SURYA PRAKASH	46	M	23875	+	-	-	+	+	-
JANAKI	64	F	87035	-	+	+	+	-	+
SOCRATES	55	M	82377	+	-	-	+	+	-
ALAGU RAJA	76	M	23587	+	+	-	-	+	-
DARWIN BRITTO	46	M	80723	-	+	-	-	-	-
GANESH PERUMAL	37	M	87235	+	-	+	-	+	-
SHAKTHIVEL	64	M	23759	-	+	+	+	+	+
ARUN MANI	57	M	87507	+	+	+	+	+	-
THANIKAIVEL	48	M	20980	-	+	+	+	-	+
AYSHA TASNEEM	64	F	27590	+	+	+	-	+	-
VEERA RAGAVAN	57	M	19897	+	+	-	+	-	-
BALU MAHENDRA	54	M	37570	+	-	-	-	+	+
KANIMOZHI	57	F	38698	-	+	+	+	+	-

VIDYASAGAR	43	M	87459	+	-	+	-	-	-
VARADHARAJ	75	M	45870	-	+	-	+	+	-
SARAVANA KUMAR	56	M	43707	+	+	+	-	-	+
BABU ANTONY	65	M	34709	+	+	-	-	+	-
KILLIVALAVAN	66	M	34987	+	+	+	+	+	-
TAMIL SELVAN	57	M	23983	-	+	+	+	+	-
ARIVALAGAN	54	M	23587	-	+	-	+	-	+
SHANKAR	46	M	25869	+	-	-	-	+	-
KARUNANIDHI	54	M	24587	-	+	+	+	-	-
VINITHA	43	F	24580	+	-	+	-	+	+
PERIYASAMY	54	M	24599	-	+	+	-	+	-
ILAYARAJA	76	M	92735	-	+	+	+	-	-
VADIVAMMAL	57	F	23586	+	+	+	+	+	-
ILANKUMARAN	68	M	76588	+	+	+	-	+	-
ASHOK KUMAR	54	M	27967	+	+	+	-	+	+
ANJALAI	68	F	58698	-	+	-	+	-	+
SOLAYAMMAL	65	F	76827	+	+	+	+	+	-
SASIKALA	64	F	43589	-	+	+	-	+	-
SUNDHARI	74	F	45986	+	+	+	+	+	-
DHANASEKAR	46	M	27097	-	+	-	-	-	+
KALAIVANI	54	F	28707	+	+	+	-	+	-
THULASIDAS	38	M	58969	+	-	-	+	+	-
THIRUMURUGAN	53	M	48888	-	+	-	-	-	-
BALAJI	47	M	83746	+	-	-	-	+	+